SUPPLEMENT

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TWELVE CASES OF INSANITY IN AUSTRALIAN ABORIGINALS WITH A COMMENTARY.

Taken from the Records of Parramatta Mental Hospital, New South Wales.

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(Continued.)

Case 6.—*Acute Mania.*

Jack, a blacktracker, aged twenty, from Eugonnia, Queensland, was admitted on December 20, 1893. His medical certificates stated that with difficulty he could be got to eat. He played with his food and threw it about. He would try to get other men's food. He was at times violent. He would fancy he was talking to some girl whom he called Lucy. He played at his cell door for hours of a night. On admission his height was 170 centimetres (five feet, seven inches) and weight 57.5 kilograms (127 pounds). He was in fairly good bodily condition with characteristic features. He was remarkably microcephalic. Mentally, he spoke remarkably good English, was shy and reticent but was not depressed, would smile when spoken to and seemed well pleased with his present quarters. He was disoriented for time, place and persons. He did not know where he was or how long he had been there. For the first night or two, he slept quietly in a single room, but when put in association he became very excited and noisy running about the ward pulling everyone out of bed. His habits were clean, he ate well, but refused to work. In February, 1894, he settled down, but refused to do any work. He spent all his day sitting about the airing court. He had no desire to leave the place, so he said. He gave no trouble. In the middle of February he began to do a little useful work and was more cheerful and had made friends with another aboriginal in the wood yard. By March 5 he looked much brighter and better in every way. He was now anxious to go out and return to his former home. On April 3 he was discharged cured. His stay in hospital was three and a half months.

Case 7.—*Dementia a Potu.*

T.D., a married man with three children, a sugar-cane worker, was admitted to the Criminal Division, 1897. He had been guilty of placing an obstruction on the railway line and had been found insane by the jury on arraignment. On admission he was found to be a big man, inclined to be stout, with the usual type of features *et cetera* of an aboriginal. His head was covered with scars. No doubt he had been a powerful man, although now past his prime. On being questioned he said that some people had given him rum and then told him to place the obstruction on the railway line. He appeared to be in a demented condition, looked dull and stupid and apparently had not even that low cunning which his countrymen as a rule acquire by constant association and contact with white men. For the first three weeks after admission there was no improvement. He was very noisy at night, did not sleep much, ate well and was clean and tidy in his habits, but still dull and would not answer when spoken to. He soon became slovenly and untidy in his habits and dress, noisy and destructive, threatening and pugnacious. During 1898 his condition remained the same, being occasionally excitable and pugnacious, assaulting fellow-patients with no provocation whatever. Apparently he had good physical health. During the next ten years, he gradually became more demented and quieter. He did not know where he was or why he had been sent there. He became simple and easily pleased, stout and in good health. He was now lazy, happy and stout and spent his time playing cards. He was transferred to Morrisset on September 9, 1911.

Case 8.—*Mania a Potu.*

Cubby, a male, aged thirty-one years, was admitted on April 4, 1890. He had been ill for fourteen days. His medical certificates stated he saw the devil staring at him. He said he was going to hunt all the white people out of his country. He was very violent and tore his clothes off. On admission he showed all the features characteristic of the Australian aboriginal, was of medium height, stout and muscular and in robust bodily health. Mentally he was excited and restless and showed all the delusions mentioned in the medical certificates. He stated that for some time previous to admission he had been drinking and since then had been troubled with the "devil." He ate and slept well and gave but little trouble. Within a fortnight he had lost his former delusions about the devil. In May he was working regularly cutting wood and was making anxious inquiries about his discharge. In July he had lost all his delusions and was quite sane. He was discharged cured on July 8. His stay in hospital was three months.

Case 9.—*Acute Melancholia.*

Nymboici Jack, a married man, aged twenty-four, from Bellinger River. On February 23, 1882, he had been convicted for the murder of his wife (an aboriginal) and had been sentenced to death. This was afterwards commuted to penal servitude for life. He was certified as insane on January 25, 1886, and was admitted to the Criminal Hospital on February 14, 1886. The medical certificates describe him thus. Very silent and instead of answering questions he bursts into tears when spoken to. He performs a number of odd and apparently purposeless actions. He has remained in this irrational condition for a number of weeks. Previously he had been cheerful, active and industrious but after being shown with some other blacks to some English visitors in the gaol, he imagined he was being identified for some further charge and suddenly became melancholic. He has completely changed, sobs and cries, is often restless at night,

is sometimes dirty in habits, cannot be got to occupy himself and appears to be in dread of something.

In the Criminal Division he made no progress but remained quiet, melancholic and dejected. On admission to the free division July 6, 1886, his height was noted as 178 centimetres (five feet, ten inches) and weight 70.75 kilograms (156 pounds). He had brightened up and now worked and showed an interest in his surroundings and was much more intelligent than the ordinary aboriginal. His mental and bodily condition rapidly improved and on July 31, 1886, he was discharged as completely cured. His history after 1886 is entirely unknown. His stay in hospital was five months in the Criminal Division and twenty-six days in the Free Division.

Case 10.—*Epileptic Insanity.*

Simon, aged about forty years, on March 3, 1877, was transferred from Gladesville where he had been admitted on December 22, 1876, for mania, cause epilepsy. On admission he was restless, suspicious and noisy by day and night and was no doubt the victim of hallucinations of hearing as well as of some delusions. It was impossible, owing to his imperfect English, to fix exactly their character and extent. Soon after admission he had a severe epileptic fit, but this did not occur again. He was at times noisy, but was never aggressive or dirty in his habits. He showed no change during 1877. He remained in good health, but was very insane, noisy and given to throwing stones. In 1878 he remained well for a few months, but developed pneumonia from which he never completely recovered, but sank and died on June 6, 1878.

Case 11.—*Dementia, General Paralysis of the Insane.*

Cumbo, a male, aged about fifty years, from Dungalear near Walgett, was admitted on March 12, 1898. On his medical certificates it was stated that he was dull and stupid, took no interest in his surroundings and was unable to answer the simplest questions although he spoke English. He would sit mumbling to himself. He required spoon feeding. He crawled about his single room most of the night. On one occasion when asked why he did this he replied that he was chasing "possums." He pulled his hair out and tried to break his arm with a boot. His habits were very faulty. On admission his height was 167 centimetres (five feet, six inches). He was in fair bodily condition, but very shaky on his legs and appeared to have some loss of power in them. (There is no record of the reaction of his pupils or of his knee-jerks). Mentally he was in a condition of dementia, quite unintelligible and unable to answer the simplest questions. Even the mention of rum and tobacco passed unrecognized. He was quiet and gave no trouble. He would wet himself during the day. He ate well and rested well at night.

By the end of May, 1898, he was feeble and demented and very helpless. In August he had bedsores on both hips and was failing rapidly. In October he was slightly better in bodily condition but helpless, faulty in habits, given to making a monotonous noise and at times destructive. In November he was then, confined to a chair and utterly demented. During 1899 there was a history of steady downhill progress. He became more and more feeble, wet and dirty and developed bed sores on the back and hips. In July he sank and died. A *post mortem* examination showed that the membranes of the brain were thickened and adherent. The brain was soft and the ventricles distended with fluid. The floors of the lateral ventricles showed small granulations like sand. The case was diagnosed as general paralysis of the insane. His stay in hospital was sixteen months.

Case 12.—*Phthisical Insanity.*

T.D., a labourer, aged forty-six, was admitted as a criminal lunatic on February 5, 1916. His crime had been entering a dwelling with intent to steal. He had been committed for trial, but had been found to be insane whilst awaiting trial. He was quite unable to give any account of himself or of his family. On admission he was found to be a full-blooded aboriginal, thin and spare, with his face much emaciated, sunken cheeks and hollow eyes. The percussion note was much impaired over the left lung in front and at the back crepitations in numbers were heard with patches of bronchial breathing on the right side. Marked bronchial breathing was heard in the infra-clavicular area and extending for some way down the chest. At the back, bronchial breathing was found at the apex and fine crepitations and crackling râles at the base. Mentally he was usually silent when addressed and did not seem to understand the questions. How much this was due to an imperfect knowledge of English it was impossible to say. He appeared scared and gazed about him furtively. His breathing became rapid and shallow. He had night sweats with but little expectoration. He rapidly became weaker and died on February 17, 1916.

No.	Sex	Age	Social Condition	Disease	Cause	Crime—if any	Cause Death	Termination	Defects—Bodily
1	F	35	Single	Congenital Mental Deficiency	Congenital	Nil	—	Unrecovered	Right cornea opaque
2	F	50	Married	Congenital Mental Deficiency	Congenital	Nil	—	Discharged	Left cornea nebulas, sight defective.
3	M	19	S.	Congenital Mental Deficiency	Congenital	Assault on boy, Sexual	Colitis	Death	
4	M	30	S.	Congenital Mental Deficiency	Congenital	Attempted rape	Phthisis	Death	
5	F	35	S.	Mania, Acute	—	Nil	—	Unrecovered	Tuberculous lungs
6	M	20	S.	Mania, Acute	—	Nil	—	Recovery	Microcephalia
7	M		M	*Dementia a Potu*	Alcohol	Placing obstruction on Railway line	—	Chronic	
8	M	31	—	*Mania a Potu*	Alcohol	Nil	—	Recovery	
9	M	24	M	Melancholia, Acute	Imprisonment	Wife murder	—	Recovery	
10	M	40	—	Epileptic Insanity	Epilepsy	—	Pneumonia	Death	
11	M	50	—	General Paralysis of Insane	Syphilis	—	G.P.I.	Death	
12	M	46	—	Phthisical Insanity	Phthisis	Entering with intent to steal	Phthisis	Death	

INSANITY IN THE AUSTRALIAN ABORIGINAL AND ITS BEARING ON THE EVOLUTION OF MENTAL DISEASE.

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From time to time the discoveries of a historic people, a King's tomb in Luxor, a city in Babylon or Cambodia, are hailed as great events in the unravelling of the past. These are, however, occurrences but as yesterday as compared to that antiquity which the aboriginal represents. Whatever may be the ultimate decision as to the age of his tribal rites and customs, he is indeed the "Grand Old Man" of the human race. Posterity will judge us adversely if we let him die without having taken his full and complete measure. The more so as his Tasmanian brother has vanished, leaving a most incomplete record behind him.

A very ingenious writer, Mr. H. G. Wells, has given the world the conception of a time machine, whereby one can travel backward through the ages. Unhappily, the instrument is not a reality, yet owing to Nature's beautiful experiment in encircling Australia with the high seas we have a similar result in that we find a race still living who resemble closely our distant ancestors of the dawn of history.

It is the custom to deplore our ignorance of this race, but until search is made through the literature we do not realize how great it is. There has not been a sufficient realization of its importance to the psychologist and psychiatrist. The student of kindred sciences has weapons or fossil remains to guide him in his researches into the past, but no calcification or mummification is possible to preserve the functions of the brain. He who would study our speciality, must use the living if possible, the alternate method of deducing behaviour from remains, utensils or weapons being of minor importance.

One of the few direct methods of attacking the evolutionary problems connected with psychiatry is by a study of the Australian black and as the years go by the opportunity lessens. Contact with civilization, phthisis and other diseases, mixed breeding and general racial decay are the order of the day. In a few years this line of research will be closed for ever. Whatever may be one's sentimental views on the passing of the primitive peoples, from the scientific or even the utilitarian aspect it will be more than unfortunate if our records are not completed before they vanish. Here is scope for either public or private enterprise to fill the gap. It must be recognized that an investigation into the psyche of the aboriginal will take considerable time. It will entail the setting up of standards, the study of the native in his own haunts and the learning of his language. Nothing less than a complete psychological-physiological examination should be the goal. It would include association tests, analysis, emotional reaction tests, intelligence quota, together with a survey of the endocrines, the vegetative and autonomic nervous systems. It will be a research of no small order, but it is long overdue. This paper is written largely in the hope that further interest may be stimulated in the matter. An attempt has been made to describe the form of the insanities of the aboriginal, to show how the colouring differs from the white, to draw inferences with regard to primitive psychology and finally to demonstrate in what respects evolution has taken place.

It is mainly based in three series of "case papers," those from Callan Park, New South Wales, and Gladesville, New South Wales, for which I am indebted to Dr. J. A. L. Wallace and Dr. H. C. MacDouall, and the third from Claremont (Western Australia) which I was myself able to collect with the permission of Dr. T. Anderson, Inspector-General of the Insane. I am also greatly indebted to Professor J. B. Cleland, both for his assistance in giving me additional material, which is acknowledged in the text and for his encouragement, without which this account would not have been written. It is easy to criticize the incompleteness of these clinical data; but, such as they are, one must be grateful for the scraps which have fallen by the wayside, to hearten the seeker of knowledge.

The Form of Insanities.

In viewing the series of cases which have been brought forward for analysis, it is necessary to stifle the wish to classify them under a modern terminology. It is a temptation to be resisted as the data is too scanty to justify the diagnosis. Although a precise definition is not possible, it is, however, justifiable to use another method of approach. This depends upon our ability to match our cases with types in a mental hospital wherein whites are accommodated. I do not think there is any doubt that this is easily possible. The admission has to be made, therefore, that there is no "new" insanity. Differences do none the less occur; the liability to pulmonary disease, the remarkable tendency to earlier dementia, the larger percentage of patients who are classified as being of destructive, violent and untidy habits. There appears to be a general tendency to motor response.

Looking at it from the viewpoint of Hughlings Jackson, one might say that the thalamic level was under poor control. Just as a child either reacts to unpleasant stimuli by an attack of hypomania, screaming and crying or by depression, sulking, so does the aboriginal lack the finer reactions. He "regresses" very easily to a lower level.

Care must be taken, however, not to generalize too freely on our series of cases. It must be remembered that an aboriginal will not be sent to a hospital without grave reason, the chief being attacks of violence. The milder cases might not come under notice. With regard to the early onset of dementia and "demented" habits, they are not in themselves evidence of a low cerebral equipment. The suggestive and imitative influences which have been operative on the aboriginal from birth, may not have tended to strict personal cleanliness and control. A strong habit of bodily hygiene or of thalamic coordination would have withstood the onslaught of a dementing process. It would be interesting to bring up the blackfellow under ideal conditions and then to notice the effect of the psychosis. Nevertheless, in default of this "acid" test, it is probable that these differences are correlated rather with primitive cerebral structure than with deficient habit formation. I have grouped the behaviour reaction in the

seventy-three cases in the series, taking as a control an equal number of consecutive cases from an old "white" case book, in which the records were of approximately equal length.

The figures show the percentage of cases in which the particular trend occurred.

TABLE I.

Behaviour	Aboriginals	Whites
Excess psycho-motor activity	60%	64%
Violence	38%	30%
Homicidal	12.5%	8%
Destructive	20%	16.5%
Dirty habits	45%	20%
Self exposure	18%	3%
Masturbation	5.5%	4%
Collecting objects, etc.	1.5%	1.5%
Hallucinations—Visual	1o%	14%
Auditory	16%	22%
(a) States of mania or excitement	63%	35%
(b) States of melancholia or depression	42%	45%
Alternating (a) and (b)	16%	12%

The increased percentage of visual hallucinations in the black is probably due to the comparative paucity of his auditory associations, lacking as he does an extensive vocabulary. This is curtailed, too, by the existence of a complicated gesture language, by which the natives communicate at a distance and at which they are very expert. The eyesight of the aboriginal tracker is proverbially acute. In classifying the cases under the heading "States of excitement" (or mania) and depression (or melancholia) there are distinct differences. The former are not relatively only in the aggregate more numerous in the aboriginal than in the white. In the latter, melancholia predominates. This finding agrees with that of Dr. Manning and Dr. Junk. It is noticed that the whites show a larger percentage who are it is considered impossible to label as being either "excited" or "depressed." This I believe to be an important difference, the significance of which is discussed later.

The Types of the Insanity.

It appears to be generally understood that in primitive races the manic-depressive psychoses are well marked entities. In this series of cases, however, there are few, that of Claremont No. 4, however, being probably an example, whilst Nos. 27 and 23 are doubtful. In No. 29 is shown well marked melancholia associated with visceral disease. No. 36 exhibits well marked phases alternating between mania and melancholia. It must not be forgotten that the early onset of dementia and the early mortality rate may well have caused the full cycle of the manic-depressive to remain hidden.

Dementia praecox is better represented. There is little doubt that the disease must be of frequent occurrence. The cases Claremont 34, 37, 26, Gladesville 1, 2, 18, could be easily matched with similar ones in a "white" hospital, diagnosed as schizophrenia. Katatonic and hebephrenic types are both represented. There is nothing like a paranoid form except in the half caste (Callan Park, No. 30). One of the patients under my personal care undoubtedly showed this disease and was in a state of terminal dementia. Dr. Jones describes another of adolescent insanity in a female at Kew. Dr. MacDouall is of the opinion that case No. 14 in his series may be one of *dementia praecox.*

My attention in Western Australia was first drawn to the question of insanity among the aboriginals with reference to their percentage of epileptics. Having been interested in recent work in connexion with protein sensitization and its bearings on epilepsy, I postulated that the influx of strange plant and animal foodstuffs to a race which had lived for decades on its original fauna should be accompanied by the increased frequency of the occurrence of this disease. To my astonishment I found that there was a history of some form of epilepsy in 30% of the cases, compared with 10% amongst the whites in the institution. This high percentage is not found in the Gladesville or Callan Park records, but it will be noted that in these latter there are a far greater percentage of old people. They are the chronicles of the last of the race around Sydney. It is fair to assume that the epileptics would die off before the series commenced. If one admits the increased frequency of epilepsy in aboriginals, at any rate in those living on the fringe of civilization, such as stations hands, we must add this disease to the long list of factors which are dooming them to extinction. It may well be noted that its importance has not been estimated properly. In this connexion protein tests done on the aboriginal might shed much light. The form of the epilepsy shows no difference from that of the whites. In the Claremont series Nos. 8, 17, 22, 24 and 33, examples will be seen of the epileptic character "troublesome and interfering," confusion and stupor. The epigastric sensation before the fit is probably demonstrated by Benban (No. 14), who felt "sullen in his stomach" and "it rose up to his head" and so he killed 'her.' The peculiar conduct sometimes seen before a seizure is shown by Hilda Mippy, No. 15, who would sing and whistle before, being afterwards excited and abusive. The way in which epileptics hang together is proverbial and in this connexion the behaviour of Benban and his friend Bilbierong is worth recording. The two were inseparable and the former would cut his forehead and also that of his chum "to make their heads better," a native custom.

Confusional insanity following pregnancy can occur. The case Claremont No. 2 showed the features of this. She made a fairly rapid and quite complete recovery.

That arterio-sclerosis and senile changes are no respectors of persons is a byword. The aboriginal is no exception to this rule. He appears to differ remarkably little in his symptoms from those of the white. The Gladesville series 3-7 and Claremont 6-7 give the main features. In them we find the shrinkage of old age, the whitening of the hair, the night restlessness, faulty habits, confusion, querulousness, delusions of persecution and states of anxiety. Taplin gives another example of an old chief who, becoming frightened, took himself to the reeds and hid away for days where he was eventually found by his friends. This senile *Wanderlust* is a serious matter in the bush and must doubtless lead to the loss of many lives. White agriculturists in the "way-back" have not infre-

quently to send their old folk to an institution for the same reason.

In a recent review in anthropology and insanity by Ales Hrdlicka, doubts are cast on the existence of general paralysis of the insane in aboriginals. There appears to be no reason for this. Dr. Jones's and Dr. Adey's patient, whose serum reacted to the Wassermann test, is clearly a case of the dementing type. A similar example is that of Gladesville No. 11, who showed marked tendencies to violence. It is unfortunate that the Wassermann test cannot be used as a criterion in others. As Dr. MacDouall states, the clinical history in his case No. 12 is suggestive of the grandoise form of the disease, but against it is the duration. In the Claremont series a diagnosis was made in cases 18-21. In the three full-bloods the onset of dementia is the most marked feature, for with it there is an entire absence of expansive ideas. This absence of a classical feature may account for the fact that the existence of disease has been questioned. One must, I think, correlate this departure from the general rule with the cerebral development. In the same way there is no evidence of anything resembling the entity paranoia. The possession of a high grade systematized delusional system is only possible with a cerebral developement more advanced than that of the aboriginal.

The occurrence of imbecility is shown by Nos. 22 and 23 of the Claremont series. The first exhibits the characteristic change of voice which is so often an accompaniment. The condition is here associated with epilepsy. The latter case unfortunately lacks a Wassermann report. It would have been interesting to have differentiated between congenital syphilitic disease and Mongolism. Mild congenital mental deficiency of the half-witted variety is seen in case No. 15 of the Gladesville records.

There remains to be described the insanities associated with alcoholism. Whatever may be one's personal view as to the case for prohibition in the "whites," no one can have any doubt that for the blackfellow alcohol is a rank poison. Tommy (Claremont No. 1) was admitted in a state of acute alcoholic mania with vivid auditory hallucinations, great excitability, dirty and destructive habits. It is remarkable how the alcoholic psychoses run true to type, that is the combination of hallucinosis and delusions of persecution associated with sex. The case of R.W. (Gladesville No. 22) exemplies this. He recovered only to return to hospital and finally died from phthisis.

The Frequency.

To attempt to give any estimate as to the frequency with which insanity occurs in the aboriginal, is impossible from these records. The older observers, who were associated at first hand with the black, varied in their accounts. Some had seen a few cases, others said that it occurred only in contact with civilization. It must be remembered that, supposing the percentage were equal to our own, a tribe of one hundred blacks might by the law of averages go for years without one of them becoming insane. Thus, owing to their nomadic habits few (if any) will be seen by one observer. As Manning pointed out, their duration of life will be small, as much owing to their savage cruelty towards those mentally affected as to their incapacity to withstand the rigors of the seasons, famine and drought.

The "Colouring."

One of the peculiarities of psychiatry, as compared with other branches of medicine is the manner in which the externals change. Although the broad structure may remain the same, owing to varying expressions, words and mode of thought, superficially an identical mental disease may appear entirely different. I have called this the "colouring of the psychosis."

Whatever may be the criticism of these series of cases as to their bearing on the form and type of the psychoses, there can be no doubt that they shed some light on the externals. One is able, by chance observations, to translate oneself back to the stone age and see them from the point of view of an alienist at that period.

Left behind are the delusions of electrical interference or of complicated deism to be replaced by the mental clothing of primitive thought.

The melancholic sees the "Devil-Devil" in the bush; he does not worry about real or imaginary sins. To the aboriginal killing has but little significance; it is commonplace. What more natural than that the dogs and "gins" should be killed because they follow or that a gin should die because she swore at her mate. Delusions of persecutions often take the form of a real pursuit, black fellows coming to chase and kill, with possibly intent to burn alive.

Snakes are common subjects of delusions or hallucinations. The best example is that of the black who with a live death adder in his hand pursued his family. One can imagine that such an individual would not be popular in his tribe nor would that other who had a habit of throwing fire sticks about in the bush.

The belief that he "could boss the blacks, because of his possessing the ulna of his father" must have connexion with aboriginal rites and customs. This may also account for the belief that the moon was his father and he was related to the sun, expressed by another patient.

I have had under my care a patient who to show his aversion to a certain society burned their books upon the pavement outside the offices. The primitive equivalent is that of "Neighbong" who "stuck-up" a station, drove the people inside with spears and then incidentally forgot about it. Delusional behaviour shows a greater tendency to be directly "motor" in the aboriginal. He does not show the same readiness to use subterfuge. There is found in other records the effect of the civilized environment upon the aboriginal and in the half-breed the primitive local colour has dropped out altogether. I have endeavoured to see into these cases evidence for the myth theory of Jung, but without success. Suggestion and imitation are in my opinion more potent than inborn thoughts and trends.

The Neuroses and Psycho-Neuroses.

The question of the stage in man's evolution during which the neuroses and hysterias *et cetera* first appeared is one of considerable interest. We have seen that certain of the psychoses exist in the aboriginal, but have discussed so far no evidence for the more superficial mental defects. Do they occur?

In the absence of treatment centres for border line aborigines, which is an impossibility, recourse must be had to induction or deduction to solve this problem. Firstly,

is their existence compatible with our estimate of the aboriginal standard of cerebral developement? The answer I consider to be in the negative. Their cerebration may be compared to the functioning of the old-fashioned printing press, which laboriously printed single copies, whereas its supplanter is infinitely more speedy. The blackfellow's thought issues slowly, whereas the average white is a quick thinker. The former seems to lack quantity of intercallary cortical neurones although it is true that visual memory centres have reached a high state of developement. So far as I have been able to find, no estimate has been made of the exact mental age of the aboriginal, in terms of the higher races, but all travellers are agreed as to the simplicity of his thought processes. The childish amusements, the inability to count beyond five, the use of only four colours, white, yellow, red, black, the absence of a future tense in verbs *et cetera*, all support this contention. His emotions as shown by his insanities are of the crude or "all or nothing" thalamic pattern. There is mania and melancholia, but the varying lights and shades and half tones of the white are missing.

How great the affective state differs from that of the white is indicated in an incident seen by Spencer. A woman was noticed to be laconically pushing the glowing end of a burning stick against the skin of her chest in order to form cicatrices. It is doubtful if the most fanatic devotee of fashion among our own race would go to equal lengths for the sake of beauty. Such behaviour is only possible in an individual whose "affect" lacks the differentiation of a high evolved type.

So far as abstract cerebration is concerned, there is a lack of "discursive operation of thought." This was well exemplified by my patient who could see no difficulties in reaching her husband in the bush, though she did not know his exact whereabouts and was dependent on the hospital for everything, even to her clothes. The primitive does as little thinking as possible; a state of affairs that is reflected in his insanities. Paranoia is unknown. It is, however, true that this factor, faculty of abstract cerebration, is not a *sine qua non* in the neuroses of the whites, as many such patients are of low mental age. To account for this I would suggest that the combination of ideation and affection is essential. In the aboriginal both are primitive, whereas the neurotic white may have only one deficient. It is perhaps too much to hope for more than a generalization as to how far he has coordinated his physiological and psychological levels, but it is certainly not impossible that structural differences in the thalamus and its relations with the cortex may be discovered. Flashman and others have proved that the latter are definitely shown in the aboriginal, though it is difficult to correlate them with precise differences in function.

The savage is a natural philosopher who says: "What will be, will be and there's an end to it." I have observed this same stoic indifference in the natives of East and West Africa.

This mental combination must be wholly unsuitable soil for the growth of anxieties, unacknowledged wishes and the like. These are usually the result of an equation between desire on the one hand and fear on the other. It is difficult to imagine a conflict when abstract desire is vestigial and fear is so crude as to show no *nuances*. Civilized races are constantly making new grounds for conflicts by their wealth of cortico-sensory associations, together with an infinite series of nicely adjusted affects. For example, the perusal of a restaurant menu card will bring realization of the number of associations and feeling tones connected with the word "food." We know that an epicure can show anger (that is retrogress or lose thalamic control) because one ingredient is missing from his *plat favorit* or a housewife can become acutely anxious over the vagaries of a new cook.

Contrast the case of the aborigine who seeing an iguana gives chase with that of a white in a similar situation. The sight of the animal was associated with the idea of food. Doubtless, it could be eaten cooked or raw as occasion demanded, but my patient's mental balance would not have been upset by the lack of salt or savoury herbs. Even a famished white might have had a conflict between desire, the result of hunger and fear of the consequences or disgust. Not so the black, for him there are two grades of food, the cooked and the uncooked: if there be food he will eat it, if not he will starve. The possibilities are extremely simple. Should the latter occur, no epic will be written, no contrast with other unfortunates will be made, nor will there be a wish to ascribe a mythical virtue or other rationalization to an enforced deprivation. As Spencer says, the native will metaphorically tighten his belt and wait until food "turns up."

This attitude is habitual towards all his "appetites"—sex included.

I cannot help thinking that an "alienist" of the stone age had a very poor *clientèle*. As if to make the occurrence of conflicts even more improbable, they are entrenched behind an extremely rigid behaviour code. The smallest events are arranged with meticulous care, they include the procedure of corroboree, ceremonies, meals, hunting and fights. It is a life of iron discipline which would be intolerable to ourselves. By the force of the herd instincts, suggestion and imitation it will seldom occur to any individual that choice is possible. Thus has life been, thus will it always be! Even the needlessly cruel and painful initiation ceremonies must appear as essential, without which the world might cease. It will as little occur to the noviciate to question his fate as for the white aspirant to civic honours on an extremely hot day to go to the hustings barefoot. It is related that on one unique occasion a black married out of his group and for this the couple were outlawed and an expedition sent to kill them. How many doubts must have been settled at their inception by the certain knowledge that justice would be of such a drastic nature. It is only when there is a possible chance of success that the extreme "tug-of-war" between desire and fear ensues. If the end be certain extinction, the latter has no opponent.

This question as to the relative importance of habit and of the association of ideas on the one hand and structure on the other lies at the bottom of most of the present day controversy in psychiatry. Is a particular morbid mental state due to a recalcitrant percept or habit or to gross change in the cerebral architecture? So far as the aboriginal is concerned, the types of insanity described are those in which cerebral structure rather than

CLAREMONT SERIES.

No.	Sex	Age	Duration	Termination	Diagnosis	Name
1	M.	40	11 months	Died, ? cause	Alcoholic Insanity	Tommy
2	F.	33	1 year	Discharged	Confusional Insanity	Louisa Haydon
3	F.	30	8 months	Died Cerebral Haemorrhage	do. do.	Kitty Yates
4	F.	20	10 years	Still Alive	Mania	Alice Mahomet
5	M.	56	1 year	Died Senility and Diarrhoea	Delusional Insanity	Jacky Jacky, alias Jimmy
6	F.	60	1 month	Died Phthisis and Fits	Dementia Senile	Lame Polly
7	M.	50	4 months	Died, ? cause	do. do.	Nooloo, alias Jackie
8	M.	30	1 year	Discharged	Epilepsy	Coolinga, alias William
9	F.	?	3 years	Died Influenza and Pneumonia	do.	Cannring, alias Darkie
10	M.	20	5 years	Still Alive	do.	Wilbing, alias Billy
11	F.	20	7 months	Died Pleurisy and Dysentery	do.	Maudie
12	M.	45	2 weeks	Died Pleurisy	do.	Jimmy
13	M.	30	2 months	Died Status Epilepticus	do.	Konkey, alias Charlie
14	M.	33	7 months	Died Phthisis	do.	Benban
15	F.	14	4 months & 5 years	Died Heart Failure	do. and Mania	Mippy Hilda
16	M.	?	6 months	Died Status Epilepticus	Epileptic Mania	Jundah
17	M.	?	2 years	Died Dysentery	do. do.	Norn, alias Johnson
18	F.	21	7 months	Died G.P.I.	General Paralysis of Insane	Cherimindi, alias Never
19	M.	43	1 month	Died G.P.I.	do. do. do.	Frank Dando
20	M.	49	2 months	Died G.P.I.	do. do. do.	Coulchakoo, alias Frank
21	M.	?	4 months	Died G.P.I.	do. do. do.	Urger, alias Banjo
22	M.	6	2 months	Died Phthisis	Imbecile (Epilepsy)	Ned
23	F.	12	6 months	Died Pneumonia	do. (Mongol ?)	Nellie
24	M.	38	4 years	Died Phthisis	Mania (Epileptiform Fits)	Bilbierong
25	F.	30	14 years	Still Alive	do.	Munyah
26	F.	20	2 years	Died Mitral Regurgitation	Mania	Grety, alias Cranky
27	M.	?	4 months	Died Pneumonia	do.	Charlie, alias Delgreen
28	M.	35	5 years	Died Phthisis	do.	Middymurringa, alias Nipper
29	M.	30	8 years	Died	Melancholia	James
					No Diagnosis	
30	M.	20	6 years	?	Prob. Dementia Praecox	Hector
31	M.	35	13 years	Died Influenza	do. do. do.	Geelong
32	M.	40	2 years	Died Phthisis	do. do. do.	Wanbin, alias Ross
33	M.	20	3 years	Died	do. do. do. (Ep.)	Piper
34	M.	24	2 years	Died	do. do. do.	Neigabong
35	M.	35	1½ years	Died Phthisis	do. do. do.	Raymuna, alias Willy
36	M.	40	11 years	Died Pneumonia	do. Mania	Koogodah
37	M.	35	2 years	Died Phthisis	do. Dementia Praecox	Dicky
38	M.	?	14 years	Still Alive	do. Terminal Dementia	Jamenta, alias Bobby

THE GLADESVILLE SERIES.

No. in Series	Sex	Age on Admis.	Duration of Stay	Termination	Diagnosis	Name
1	M.	youth	1 year	Died Phthisis	Dementia (Primary	Frank Tivoli
2	M.	34	5 days	Died Phthisis	Dementia	Jack (1)
3	M.	102	6 months	Died Bronchitis	do. (Senile)	Tullymorgan Dick
4	F.	70	2 months	Died Dysentery	do. do.	Mary Curall
5	F.	65	3 months	Died Phthisis	do. do.	Mary Billagong
6	M.	?	8 days	Died Senility	do. do.	Boney
7	M.	50	9 months	Died Brain disease	do. (Senile ?)	Dick
8	M.	?	2 years	Died Pneumonia	General Insanity	Charlie
9	M.	50	3 months	Died Brain disease	General Paralysis of Insane (prob.) and Acute Mania	Billy Murray
10	M.	55	2 years	Died G.P.I.	General Paralysis of Insane	Bucklebone Jack
11	M.	50	8 months	Died G.P.I.	do. do. do.	Henry Bob
12	M.	40	6 months	Died G.P.I.	do. do. do. (prob.)	Bumbang Jack
13	M.	70 ?	1 year	Died G.P.I.	do. do. do.	Jimmy Combo
14	M.	25	4 years	Still alive	Imbecility without Epilepsy. Dementia Praecox ?	J.C.
15	F.	60	8 years	Died Cardiac syncope	Imbecility(congenital) with Delusional Insanity	S.A.
16	M.	?	6 years	Died Brain disease	Mania, Acute	Tommy
17	F.	40	6 months	Died Phthisis	do. do.	A.H.
18	F.	40	3½ years	Died Diarrhoea	do. Sub-Acute	Eliza (2)
19	F.	50	9 years	Died Diarrhoea	do. Delusional	Betty C (3)
20	M.	50	6 months	Discharged	do. do.	Fred
21	M.	60	6 months	Died Acute Encephalitis	do. do.	Mumbulla John
22	M.	39	7 years	Died Phthisis	do. à Potu	R.W.
23	F.	50	1 month	Discharged	do. (prob. à Potu)	Sal. W.
24	F.	30	1 year	Discharged	do. Delusion al (à Potu)	Matilda
25	M.	60	2 weeks	Died Senile Mania	do. Senile	Charlie
26	M.	50	4 months	Died Pneumonia	do. Delusional, with Senile Dementia	Pointer

(1) See Callan Park and Parramatta Records.
(2) Parramatta Records.
(3) „ Parramatta Records.

function is involved, such as epilepsy, senility, the psychoses *et cetera*. There is little evidence that the others occur. Is this freedom due to the restriction of conflict rather by reason of individual discipline than to a primitive cerebral mechanism? There is distinct evidence to the contrary. It so happens that in every mental hospital the ingrafting of a neurosis upon a psychosis is a daily occurrence. The individual desire for freedom which is accompanied with a dread lest the incarceration should be permanent, results often in a state of anxiety, familiarly termed "the prison complex." The medical officer is asked many times a day by patients to be set free. Others react more indirectly by querulousness, abuse or exaggerated emotional traits.

An attempt has been made to contrast the number showing this behaviour, taking as control an equal consecutive series of whites.

TABLE II.

Behaviour	Aboriginals	Whites
The prison complex	5%	17.5%
Suicidal trends	0	8%

It is fair to surmise that the aboriginals of the groups under review had practically all come under the influence of civilization. The authority of Totem and Tabu no longer held them bound in the same meshes of inflexible tradition. Thus, they might have been expected to produce a few anxiety states, but, of the 5% enumerated, two-thirds were in half-castes and the other a case of senile dementia. A further example of the same type has been described by Carr, but undoubtedly fear in the aged is a part of a psychosis and not evidence of a neurosis.

The only feasible explanation to my mind of this immunity is that it depends on the primitive nature of the cerebrum rather than the mode of life. It is not the "idea" itself, but the structure beneath that matters. The wish to commit suicide is one of the hall marks of the neurotic mind. It is remarkable that the control series of cases show 8% in which this tendency is mentioned, whereas it is absent in the blacks.

The worried psychopath of to-day might well be forgiven if he looked with longing upon an evolutionary stage, in which psychæsthenia and neurasthenia were non-existent. Civilization has gained much in the aggregate, but the individual sometimes loses. It is Nature's way.

Conclusion.

There is evidence that the aboriginal shows the following characteristics in his insanities.

1. An early onset of dementia, increased tendency to motor, trends, to visual rather than auditory hallucinations.

2. The paranoid types are not represented.

3. *Dementia praecox* and manic-depressive insanity occur.

4. Epilepsy is probably more frequent in those who are in contact with civilization, and may be a factor in the racial extinction.

5. Confusional insanity and that associated with senile changes and alcoholism differ remarkably little from those in the whites.

6. General paralysis of the insane is not infrequent and exhibits the demented form.

7. The aboriginal has not reached an evolutionary stage in which the neuroses and certain of the psycho-neuroses, Hysteria and the phobias can exist.

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Section IX.—Diseases of Children.

AN ADDRESS.

By R. B. Wade, M.D., Ch.M. (Sydney),
President of the Section; Honorary Surgeon, Royal Alexandra Hospital for Children, Sydney.

In opening the proceedings of this Section of Pædiatrics, it is my first and most pleasant duty to extend a hearty welcome to all of you who are interested in this branch of medicine.

Though this Section is one of the last to be established in Congresses in Australia, it is gratifying to see that the number of men interested in this Section is increasing.

A point worthy of notice is the trend of University opinion towards making the study of diseases of children a separate and compulsory part of the medical curriculum both here and in the old country and the University of Melbourne is to be congratulated on the advanced position it holds in this respect.

Apart from the large amount of work that the general practitioner has to do amongst children, roughly about two-thirds of his total work, which requires a special training in the examination of children and the diagnosis of conditions like the developmental and nutritional diseases that are peculiar to early life, of conditions that differ in many respects in their clinical manifestations from those of the adult, the greatest scope of the pædiatrician must and will be in his effort along the lines of

preventive medicine, for surely he of all men has the greatest knowledge to direct human life along the right paths and most appropriate lines to insure a strong healthy nationhood both mental and physical.

We need only look around to find the various interests that are at work on the question of improving the mental and physical status of the child, most unfortunately dealing with the afflicted child, whether it be from nutritional disturbance or from mental deficiency, and the rest that are trying to help to build up the healthy nationhood. It is a striking commentary on our civilization to see the way that money is to spare for the enjoyment of the doubtful education of the American picture show, but none—or but little—to improve the nation's physique; these overworked bodies have two things in common, first their earnest effort to do something for the benefit of child life and lastly their common state of penury.

These varied and various interests are all working on their own separate lines, all good in their own way; one little realizes how much really good is done by these whole hearted small bodies of workers and givers, all hampered and limited in their scope by paucity of funds and in this last respect most of all are those financed and managed by Governments.

Let me mention a few of these activities to show their wide scope and possibilities; baby bonus, State children's relief departments, mental deficients asylums, State education and State education for backward children (still in its infancy), baby clinics and medical inspection and dentistry for State school children; to mention some of the Government interests and of the purely charitable, the milk depôts, hospitals, denominational homes for orphaned children.

All these are working more or less apart and though many of them are working well and along good lines, they are as yet unable, owing to lack of funds, to cope thoroughly with their object and many of them are at cross purposes. A greater recognition is taking place of the needs of the child and the possibilities to be achieved, but we are still only on the threshold and have yet to realize what can be done to build up through the children, a sturdy, alert nationhood, strong both mentally and physically. One need not stress the point that there is need for larger funds and most of all for some settled scheme of central control that will prevent overlapping and direct these various organizations along lines of greater cooperation and coordination.

While on this subject I may note what a field of possibilities is open to us when the functions of the endocrine glands become an open page and endocrine meditation is no longer a matter of guess work and generally a failure.

It is with deep and heartfelt regret that we must record the death of the last President of this Section, the late Dr. Litchfield; a man of wide reading and unusually clear reasoning powers, he was in past congresses a tower of strength to our section. We shall all feel the loss of his instructive and kindly criticism and comment on the matters under discussion.

In conclusion, I should like to tender my grateful thanks for the honour that has been done me in electing me President of the Section. I feel by my separation to the surgical side of things, I lack the very necessary knowledge to fill the position as should be done, but know that I shall have your cordial assistance to help me with the work of the Section. I am sure you will find, thanks to the zeal and untiring work of your Secretaries of Section, that an interesting programme has been put forward, one which will insure that at the end of the session much interesting discussion has been secured and that we shall all feel the benefit of having heard the other man's or woman's views on our pædiatric problems.

I have much pleasure in declaring this session open.

PYLORIC STENOSIS.

By Stewart W. Ferguson, M.D., B.S. (Melbourne),
Honorary Physician, Children's Hospital, Melbourne.

In opening the discussion on congenital pyloric stenosis I do not propose to give an account of the classical symptoms of the condition, but rather to sketch the management of such cases as we meet them in the wards of the hospital or in private practice. In a recent article Still said that all babies who showed vomiting, constipation and failure to thrive should be regarded as possibly suffering from pyloric stenosis. With this dictum I am heartily in accord. Every case by no means possesses all the typical symptoms, but if we meet with this condition it is our duty to exclude the possibility of stenosis. The palpation of the tumour is often very difficult, sometimes impossible without an anæsthetic and one often has to be very patient in one's examination before perstaltic waves can be seen.

It must, however, be remembered that there are very many patients who present the typical symptoms of persistent vomiting, constipation and failure to thrive and imitating in every way the manifestations of a typical case, except that no tumour can be palpated. Irregular feedings or too large feedings or ill-balanced feedings can produce a condition of irritability of the stomach which closely simulates in symptomatology pyloric stenosis. This condition can readily be cured by proper modification of the size and nature of the feedings and regulation of time of feeding.

Coming now to the investigation of cases of supposed stenosis, it has been my routine practice in every case of suspected stenosis to examine the patient with a bismuth meal and the findings are in every case quite characteristic. The meal is given any time from six to twelve hours before the screen examination and consists of eight grammes of barium sulphate.

The first thing noticed is that practically all the barium remains in the stomach; often twelve hours after the meal has been taken none has left the stomach, though usually if the examination is made as late after the ingestion of the meal as this, there is a little in the intestines. It is usually seen as a rounded mass lying in the lowermost portion of the stomach well below the level of the umbilicus; sometimes, however, it is more elongated and lies spread out along the greater curvature. There is no evidence whatever of stomach tone. The child is now slowly revolved so as to lie on its right side. The barium with the change of posture slips over as if greased and remains the same apparently inert mass lying over the

pyloric orifice and no peristalsis whatever can be demonstrated. Furthermore its position is important as it lies almost in contact with the right side of the abdomen. The rotary movement of the child is continued slowly so that it is made to revolve in a complete circle. The main factors noted are the considerable activity with which the barium moves, and the evidence of dilatation of the stomach. When the child lies on the left side the barium again rests practically on the left side of the abdomen. The stomach, measured by the barium excursion seeming to extend almost from one side to the other.

Unfortunately my controls have never been quite healthy babies, but were children admitted into the hospital with some other disease. Some were convalescent from pneumonia, but the majority were babies admitted with some other form of nutritional disturbance, at any rate they were not perfectly healthy babies, otherwise the contrast might have been greater.

The main points of difference in the screen examination are as follows: In both the barium lies as a bolus at the bottom of the stomach. In the stenosis babies the barium very rapidly and quickly reacts to gravity, while in the other babies it moves much more slowly and sedately to the lowermost points according to the position of the patient. In the stenosis babies there is the evidence of gross dilatation of the stomach which was absent in the normal baby. Also in the stenosis babies the barium lies inert over the pyloric orifice, while in the controls the peristalsis can be seen and small pieces of barium are bitten off and can be seen leaving the stomach. In the controls the screen examination was undertaken one hour after ingestion of the meal, so that not only do we have the main evidence of the retention of the barium in the stomach, but also other very characteristic findings.

I use these findings from the bismuth meal investigation for other purposes than mere diagnosis. We get definite indications for the method of treatment, surgical or medicine.

If at any time after six hours there is practically complete retention of barium in the stomach and if this is supplemented by the other findings enumerated, I have no hesitation whatever in recommending immediate operation, no matter what the condition of the baby is. If ill-nourished or marasmic I do not think anything is gained by delay. The stomach should be washed out and the baby fed on sugar and water while the necessary arrangements are being made.

Another baby with a different type of affection in whom vomiting was a prominent feature, weighed five kilograms (eleven pounds) and was not constipated. It appeared healthy and I did not regard its condition clinically as a stenosis. However with X-ray examination it gave characteristic findings. He was operated on by Dr. Downes with the happiest results. The stenosis was extreme. Even if this baby might have recovered with medical treatment, as appeared likely from the clinical history, it was saved months of treatment and the likelihood of increasing dilatation of the stomach. The results of surgical intervention are so often so dramatically successful that having definitely satisfied myself with the diagnosis of congenital pyloric stenosis, I unhesitatingly recommend operation to be performed as soon as reasonably practicable.

I am, of course, referring to the Rammstedt operation which in capable hands entails little shock and can be rapidly performed. The only failures I have seen are in babies weighing under two and a quarter kilograms (five pounds) in whom the condition has not been recognized till too late and in whom there is very little resisting powers left. Even in these I would recommend operation, as I consider it furnishes less risk than the continuation of medical treatment.

The barium meal examination reveals yet another type of case. Some babies when examined six hours after ingestion of the meal, show a considerable quantity passed into the small intestine. I find it very difficult to estimate relative amounts retained in the stomach and amounts in the bowel. In the former it remains a solid bolus, while in the intestine it is scattered all over the place. If, however, notwithstanding undue retention in the stomach there is a fair amount in the bowel, then the babies should be treated medically. I have regarded the one series as definitely and organically stenosed and of congenital origin and the other as obstruction due to pyloric spasm. Dilatation is never so extreme in these cases and I have never palpated a tumour. This division may be too arbitrary, but at any rate it furnishes a good working basis from the point of view of treatment.

The essential treatment of such cases is dietetic and I think there are in this respect two main rules to be observed. Firstly, the food must be of such a nature as to remain fluid in the stomach and so pass more readily through the pylorus. Secondly, the fat content of the food should be kept low. The higher the percentage of fat in a feeding, the longer is such food retained in the stomach and hence the increased liability to vomit. We know that fat, though desirable, is not an essential in the diet of infants, so that it can safely be kept at a very low percentage for a long period without harm resulting. As fat is low and caloric needs are high, such infants must have a relatively high carbo-hydrate content. There is no difficulty in maintaining this in the majority of babies, provided we give varied carbohydrates. My usual practice is at the outset to discard all milk mixtures. While various diluents as whey and barley water may delay rennin clotting, there is always a certain amount which must mechanically interfere with the rapidity of the passage of the food through the pylorus. I used to use skimmed milk and whey mixtures, but have discarded them in preference to the patent foods. Such foods as "Glaxo" and "Lactogen" I have also discarded for this purpose owing to their high fat contents. Condensed milk and "Horlick's Malted Milk" with their practically negligible fat contents, their easily digested, dessicated protein which does not form a clotted mass with rennin, and with their high carbo-hydrate content furnish much more the type of food indicated. A mixture of the two foods is often useful, especially if the infant shows a cane sugar or a dextrin-maltose intolerance. With the carbo-hydrates mixed in this way, they will often tolerate a much higher sugar percentage. After

a time, however, it may be found that these infants may fail to thrive. The relative amounts of sugar and protein in these foods are so out of proportion that a continuance of such a diet must result in protein starvation and consequent impaired growth. Artificially fed infants must have a higher percentage of protein than breast fed. This is because those amino acids concerned in growth and development of the human infant are derived mainly from the lactalbumin. As casein is the predominant protein in all infants' food, more of it must be given to supply a sufficiency of these amino acids. This deficiency of protein I supply by the use of "Plasmon." This is first converted into a jelly and a necessary amount to produce the desired percentage of protein may be dissolved in the feeding.

On such a food mixture *plus* added vitamins the child will thrive readily. Furthermore such a feeding *minus* the "Plasmon" can be used as a vehicle to which milk can be slowly and gradually added as the child improves. I say *minus* the "Plasmon," because such children will not often tolerate the fat that is being added with the milk, if the protein percentage is too high.

So far I have only spoken of the principles of feeding. It goes without saying that regularity of feeding is an essential. If the baby is small and frail, I prefer small feedings at interval of two to two and a half hours; if in fair condition at intervals of three hours. The size of the feeding should not be too large. Another method of feeding these infants is to thicken the meal with a cereal, the idea being that the thickened feeding is less liable to be vomited. Personally I have derived no benefit from this method and I also think it is wrong in principle. I aim more at maintaining the fluidity of a feeding, not the reverse; to give such a child large quantities of starch is obviously a dietetic error. It already has sufficient to contend against without adding an excessive quantity of starch to its burden.

Drugs to my mind are of much lesser importance in the treatment of the condition.

One American observer who did a series of test meals in these cases, found they were all associated with excess of free hydrochloric acid. He also found that sodium citrate was the most satisfactory drug in neutralizing this excess. I have generally added it to my feedings as a routine.

As regards antispasmodics I must confess to being very disappointed with any help I have derived from them. I have used mainly opium in small doses before each feeding and also belladonna, but with little or no satisfaction.

Lastly, I mention the well established practice of washing out the stomach. I rarely practise it now by routine. If there is much retention in the stomach, I think an occasional wash-out three hours after a meal and the giving of the subsequent meal through the tube is sometimes of value. It is, I think, of more value in treating those babies in whom milk mixtures are persisted in as the method of feeding. It removes retained curd.

In conclusion the main point I would like to emphasize is the importance of the early recognition of the condition and the institution of appropriate treatment.

CONGENITAL PYLORIC STENOSIS.

By RUPERT M. DOWNES, C.M.G., M.D., M.S. (Melbourne),
Honorary Surgeon, Children's Hospital, Melbourne.

Dr. Ferguson has laid stress on certain phenomena that he notices on screen examination as diagnostic of pyloric stenosis. Other men rely on other clinical features. The most important point, however, in the diagnosis is to have in one's mind the possibility of pyloric stenosis in any young infant that one is called on to treat which has a history of persistent vomiting. Just let there be that suspicion and the diagnosis is comparatively simple. I speak feelingly as recently I have had several babies that died after operation who had been under observation so long without a correct diagnosis being made that they were too feeble and emaciated to survive the shock of operation. When one has come across several patients who have been treated for weeks by practitioners and even pædiatricians who have failed to notice the pyloric stenosis that has been recognized at once by the hospital resident medical officer, it is apparent that it is not generally realized that this condition is by no means rare. Probably among the many babies that die from marasmus, gastritis, dietetic causes and the like, there are some that are really subjects of pyloric stenosis. Doubtless among many cases of vomiting infants, if the mother does not give a clear history or if the examination be made when no peristaltic waves are showing or no abdominal examination is made at all, it is not easy to conceive the possibility of pyloric stenosis. Let me say that I speak merely as a surgeon who only sees a patient when the diagnosis has been made or at least queried and so have no real appreciation of the ease or difficulty of primary diagnosis.

With regard to the signs on which a positive diagnosis is made, I am not satisfied until the typical tumour has been felt. Generally this is easy if a little patience be exercised, but if the stomach be dilated, it may be impossible, even under an anæsthetic. One failure under such conditions is insufficient and there should be no hesitation in giving an anæsthetic if the muscular resistance cannot be overcome otherwise. In every baby on whom I have operated, the tumour has been palpated and recognized prior to operation. The majority of surgeons, I think, are satisfied to diagnose the condition if well marked peristaltic waves be observed, but I have seen one infant in whom the waves were extremely prominent and the only lesion found *post mortem* was a severe intestinal inflammation. X-ray examination is, I consider, rather an unnecessary proceeding and a method of interest only. It has the disadvantage that the patient is often considerably knocked out by it. One observer writes of a typical rhythmic snake-like contraction of the pylorus, seen immediately after a barium meal, which he considers pathognomonic. I have tried, but failed to observe this phenomena. Another bases his diagnosis of spasm or stenosis on the relative proportion of barium in the stomach or intestines four hours after the meal. This does not appear very sound or exact to me. As far as I have observed, it is unusual to find a delay of six hours in any condition except a stenosis, but quite re-

cently I have seen a baby not suffering from pyloric stenosis in whom a complete delay of eleven and a half hours was observed.

Dr. Ferguson's observations are extremely interesting, especially in relation to spasm of the pylorus in which a tumour apparently is never found, but if a tumour can be felt, as it always can in a true case of stenosis, why add the fatigue and expense of an opaque meal examination?

Considerable difference of opinion has existed with regard to the relative merits of medical and surgical treatment and statistics of results have been freely advanced in favour of one or other method. The general trend of opinion at present seems to be in favour of operation. One must be largely guided by one's own experience and it is remarkable that in the Children's Hospital, Melbourne, no case is on record in the last three years in which the infant has recovered under medical treatment. A large majority of those treated by operation on the other hand have been cured. I am of opinion that if a rule of thumb is to be followed, it would be far better to operate in all patients than not at all and that very few operations would be unsuccessful if the correct diagnosis were made soon after the onset of symptoms. Yet I think it is justifiable to give a trial to medical treatment if the infant be in good condition when first seen. If there be no definite improvement within a week or if the loss of weight be rapid at once, then operation should not be delayed. When a baby is wasted and in poor condition, it appears that operation is its only chance. No baby is too ill to be given this chance. The baby in the most apparently hopeless condition that I have seen, recovered after operation. Pre-operative injections of saline solutions intraperitoneally (though this makes handling of the pylorus more difficult) or subcutaneously with 1% or 2% dextrose added should be given in badly wasted patients.

Close attention to detail in the operation is necessary to obtain good results. The Rammstedt operation of simple longitudinal section of the muscle coat of the pylorus, which is the only one of which I have had any experience, is quite simple to perform. Bad though the practice be of operating by the clock, this is an operation that should be performed as rapidly as possible, though, of course, with gentle handling of the pylorus. To further this the few instruments required should be close to hand, sutures threaded beforehand and packs and dressings ready.

Heat is very necessary to diminish shock. It is advisable to have the child lying on a hot water bag during the operation and it should be put into a hot-air bed immediately after operation. A hot-air bed is readily improvized by covering an electric bulb with blankets supported on a cradle.

The other important point in after treatment is not to starve the patient. He may be considered as a starving infant in whom the cause of the starvation has been removed and he requires good food in small quantities as soon as possible. His chance of life is much better if he can be put on breast milk a few hours after operation. Failing that, equal parts of milk and water or condensed milk is advisable.

The critical time is the first forty-eight hours after operation. If he survive that, his chances are good.

Dr. T. E. Green said that although he had not had a very large experience in pyloric stenosis, he had seen three babies affected in this manner successfully treated by gastric lavage, careful dieting and the exhibition of small doses of tincture of opium before feeding.

Dr. Gilbert Brown emphasized the importance of careful selection and administration of the anæsthetic in surgical measures for the relief of pyloric stenosis. He advocated the injection of 0.3 milligrammes of atropine as a preliminary to the anæsthetic, induction of anæsthesia with ethyl chloride and continuance with ether delivered by the open method along with oxygen through a Shipway apparatus. The ether was administered through a nasal catheter.

Dr. H. Gilbert said that he had seen recurrence of symptoms after the Rammstedt operation in spite of the fact that there was decided immediate improvement which persisted for some days. The recurrent symptoms subsided slowly under careful dietetic management.

Dr. J. Macdonald Gill advised persevering with atropine as medicinal treatment. A dose of 0.06 cubic centimetre of a 0.1% solution might be given in each bottle and the amount of atropine increased daily by 0.06 cubic centimetre of the solution until a daily quantity of one cubic centimetre was reached or the vomiting ceased. Such measures had been found useful in vomiting due to causes other than pyloric stenosis. With reference to diagnosis Dr. Macdonald Gill said that he regarded the appearance of peristaltic waves after feeding as almost decisive even when no tumour could be palpated.

Dr. H. Douglas Stephens said that he had discarded medical treatment in the condition under discussion in favour of the unmodified Rammstedt operation. He did not utilize X-ray examination frequently in diagnosis and considered that it was often unnecessary.

Dr. Truby King remarked that diagnosis of pyloric stenosis was often difficult and required great patience. It was better to operate too soon than too late. In the post-operative feeding breast milk was to be chosen before all else; failing breast milk they should strive for a mixture or modification of cow's milk which closely approximated human milk in the proportions of its constituents.

Dr. R. B. Wade drew attention to the difference in the results obtained in the treatment of hospital and private patients and referred to a recent paper by Dr. G. F. Still on the subject of pyloric stenosis. He frequently found the tumour difficult of palpation and relied chiefly on the presence of peristaltic waves. In the case of pyloro-spasm the onset of the vomiting was usually at a later date than that which appeared as a symptom of pyloric stenosis. Pyloro-spasm was often cured by the gastric dilatation developed.

In clinical teaching all students should be thoroughly apprised of the seriousness and importance of pyloric stenosis and in treatment surgical measures should be adopted as a routine.

Dr. S. W. Ferguson in reply reiterated his views on the importance of "high protein" feeding in the post-operative management. In any discussion as to the relative merits of medical and surgical treatment, it was to be remembered that two different conditions had to be considered. One, congenital hypertrophy of the pylorus, required surgical measures and the other, pyloro-spasm, often responded to medical methods.

DIAGNOSIS AND TREATMENT OF TUBERCULOUS CERVICAL GLANDS.

By R. M. Downes, C.M.G., M.D., M.S. (Melbourne),
Honorary Surgeon, Children's Hospital, Melbourne.

I wish to express my appreciation of the honour you have done me in inviting me to open the discussion on tuberculous cervical glands. Through the kindness of my colleagues I have had for some time the care of all patients with this disease coming to the Melbourne Children's Hospital. Their number is strikingly less than it was some ten or twelve years ago, perhaps owing to the present day frequency of enucleation of the tonsils.

The cervical glands form the great majority of external lymphatic glands affected with tuberculosis. The type of organism appears to vary, but in children it is usually of the bovine variety. The disease is said to be most frequent in the second decade, but in my experience it is more common at an earlier age. In many cases the tonsil is tuberculous, the glandular affection being secondary to it.

The proportion of associated tuberculous tonsils reported ranges from 13% to 75%. In twenty-one of my patients examined the tonsils were tuberculous in 43%. It is not uncommon too to find associated tuberculosis of the post-nasal adenoids. The mastoid antrum and dental pulp are also occasional primary foci. Fraser points out that the limitation of tuberculosis, secondary to any of these foci, in a localized group of glands forms a point of difference from all other forms of tuberculosis. In such cases the tubercles are found chiefly in the cortical follicles, in contra-distinction to the type due to blood infection in which the affected glands are scattered throughout both sides of the neck and the tubercles are central. The latter type appears to be rare in this country. A third type is described in which the infection spreads from the mesenteric glands.

Diagnosis.

It is important to make a diagnosis as early as possible, before the skin is involved. This is often a very difficult matter. It is usually stated that the disease is chronic and rarely or never acute in onset. My experience does not confirm this, for I have seen a number of patients in whom an abscess has been the first sign noticed. Not infrequently these abscesses are opened and only their failure to heal and the formation of the typical pale-œdematous granulation tissue around the sinus leads to the recognition of their true nature.

It is safer to regard every abscess of a cervical gland as possibly tuberculous. If no primary septic focus be found, tuberculosis becomes more probable. The von Pirquet test gives some help, a reaction being conclusive in infants and very suggestive in children under six years of age. The absence of a reaction is strongly against tuberculosis.

It is perhaps more difficult to decide as to the diagnosis of tuberculosis in many cases of enlarged glands that present no signs of softening. Even to decide whether the glands are enlarged or not is far from simple, for there are very few children in a hospital clinic in whom they cannot be palpated. One child whom I have under observation, when first seen presented glands that gave no suspicion of tuberculosis or even enlargement. Examination of her tonsils after their removal, showed numerous tuberculous foci. Following this the glands enlarged considerably and then became quite hard, presenting a picture that would be classed as tuberculous on clinical grounds alone. Other cases have been met in which the discovery of the tuberculous nature of the glands has been quite accidental and unexpected. In most cases, however, the difficulty arises in the diagnosis of definitely enlarged glands. Examination of the enucleated tonsils may help, for if the tonsils be tuberculous, there is a probability that the enlarged glands have been infected theretrom; at any rate it is safer to consider them as tuberculous. In many cases the greatest information will be obtained by watching the course of the glands after any septic focus has been removed. Subsidence is evidence against tuberculosis, but it must not be forgotten that a history of repeated alternative swelling and subsidence is not uncommon in children who are eventually shown to be tuberculous. Perhaps the initial inflammations are pyogenic, the tuberculosis secondary; perhaps they are concomitant.

No hard and fast rules can be laid down for the diagnosis of tuberculous glands. There are few diseases in which a definite diagnosis may be harder. It is largely a matter of experience that enables one to judge rightly, but I am led to believe that far more enlarged cervical glands are tuberculous than are so diagnosed.

Treatment.

In Australia the prognosis as regards life is good under any form of treatment. Our chief aim, therefore, is to decide what treatment will give the best and quickest result with the least damage in any individual case. We may consider the following methods: (i.) general hygienic or expectant treatment, (ii.) the administration of tuberculin, (iii.) surgical treatment and (iv.) X-ray treatment.

While we can discuss treatment under these separate headings, it must be realized that we employ several or all of them in most cases. One is struck by the general preponderance of opinion in favour of radical operation shown in the discussion on the subject at the 1922 annual meeting of the British Medical Association. That is the aspect I propose chiefly to consider.

It does not follow that because results under conservative treatment in the United Kingdom are less successful than after operation, that it should be so in this country. Conditions are not similar. Here we have more sunlight, a better supply of food, less overcrowding and a more open air life; all in favour of resistance to the tubercle bacillus. We hope, too, that our milk is more free from that bacillus.

General hygienic treatment entails good, well cooked food, fresh air, sound sleep, amusement, avoidance of fatigue and boredom. Heliotherapy is a potent factor for good.

Tuberculin, I think, is not regarded by the bulk of the medical profession as of very great value. Some express themselves enthusiastically in favour of it. Personally I have not had any very striking results with it, but I certainly think it is of some limited value and make use of it in most cases. The form used in the Children's Hos-

pital, Melbourne, is the bacillary emulsion. The initial dose used is 0.0001 milligramme with gradual increase, at first in bi-weekly then in weekly doses until a local infiltration occurs. In many cases when the dose reaches 0.01 milligramme or thereabouts small abscesses, slow to heal, form at the sites of injection. Lately I have been using an inunction of 10% to 25% old tuberculin, as recommended by Phillips, which is rubbed in by the gloved finger once a week in the axilla or groin. It is followed by an unimportant skin reaction which may persist for several days. This method has the advantage of avoiding the needle punctures which is a gain in small and nervous children.

Turning to surgical treatment it may first be asked: "Does any treatment short of complete excision cure the disease?" The answer will generally be in the affirmative, but Fraser and others express considerable doubt. It requires long experience to speak with authority on such a question, but I cannot help thinking that as so many tuberculous glands with little signs are encountered, sometimes almost accidentally, there must be some children who grow up with the disease unrecognized and show no further signs in adult life. Furthermore the calcification sometimes found in mesenteric and cervical glands appears to betoken cure. Then there seems no reason why, if cure takes place in other tissues, of which there is little doubt, it should not occur in the cervical glands. If the interpretation of the frequent reactions to the von Pirquet test as being due to a healed tuberculous lesion is correct, it adds weight to the possibility of natural cure.

In deciding whether to employ surgical treatment and how, the different types of cases must be considered. The most difficult is one in which in the upper portion of one or other internal jugular group there is a collection of enlarged discrete glands of a fleshy consistency, not tender, forming a distinct swelling in the neck. If one wants to remove these glands easily and with the prospect of a faint scar, now is the time to do it, before there are softening and adhesion to surrounding structures, which involve the probability of bursting the caseous mass during removal.

What are the reasons against excision in such a case? There is the risk of operation, which is little more than that of the anæsthetic, and there is the mental disturbance to parents and patient, arising from the anticipation of operation and the natural human dread of the knife. Of more importance is the possibility of dissemination of the tuberculosis as a result of manipulation. I cannot myself recollect a case in which this has happened and think that the risk can be almost ignored.

To my mind the chief drawback is the operation scar, especially in a girl. This is no small matter, for it is in a situation in which it can always be seen and a visible scar on the neck is a decided drawback. The mother at the outset, filled with alarm as to the whole condition, will express her indifference to such a small thing as a scar, but when the fright and the glands are gone, she will be much more concerned about it.

Then again it is said that one cannot be sure of removing all the affected glands and that is so; but in practice it usually is found that a free removal of all the obviously affected glands is quite effective.

What are the arguments in favour of excision? The chief one is based on the conception that in the great majority of cases the disease is purely local and that the glandular infection results from a primary focus drained by the infected glands. If the infected glands and the primary focus be removed, cure is rapidly attained. The time factor may be a very important matter, when frequent visits to hospital involve the question of the parents' wages or the child's education. The second reason in favour of excision is the diminished likelihood of a poor cosmetic result. Holt says that 50% of these cases suppurate. The trouble is that one cannot foretell in any particular case whether the glands will break down or not. And when they do the process is sometimes very rapid. It is not uncommon for glands which appear to be progressing satisfactorily, suddenly to become soft and in a very few days to lead to invasion of the skin before anything can be done to save it. The result is a sinus which on healing produces a scar far more unsightly than the narrow linear operation scar of early excision, the opportunity for which has been lost.

Fraser's blood infection class however is said not to lend itself to excision, recurrence being the rule.

My own view is that conservative treatment may be adopted only if the child's general living conditions be satisfactory and if he can remain under regular and frequent observation, so that the earliest appearance of suppuration may be detected. Failing these conditions or if after some months there is no sign of improvement or at any time the mass becomes fluctuant, then excision should be carried out. In any case the primary focus should be removed at once. As a result of the investigations mentioned I am very strongly of opinion that the first thing to do in all cases of tuberculous cervical glands is to enucleate the tonsils and clear out the post-nasal growths. Though the tuberculous tonsil cannot as a rule be detected by ordinary clinical examination, I have noticed that it often has a characteristic appearance, that may be pictured as that of an anæmic mulberry. In addition the mastoid antrum and carious teeth, if showing signs of disease, should receive appropriate attention.

In regard to the operation of excision the scar is less noticeable if the excision be made transversely or obliquely, exactly following one of the natural folds of the neck. Though this renders the actual operation more difficult than if the more usual incision along the anterior border of the sterno-mastoid be made, I think the improved cosmetic result warrants it. I use Michel's clips to close the wound, removing them on the second or third day.

The next class of case is one in which the glandular mass is fluctuant. The chief object here is to prevent infiltration of the skin and resultant sinus formation. The commonest treatment is to aspirate the abscess contents and to inject some variety of iodoform emulsion. Aspiration, however, is not always as easy as it sounds. The caseous contents require specially made large bore needles and even with them the procedure may be difficult. The process generally has to be repeated and to small children is a terrifying performance. Parents too

do not take kindly to repeated operations. My own practice is to aspirate once or twice, if necessary, as a rule washing out the abscess cavity through the needles with "Eusol" which I have found sometimes to give satisfactory results, though I have no great faith in local antiseptic applications. If there is any reddening of the skin, it is important to make the needle puncture through healthy skin, which heals better. Should fluctuation appear after the second aspiration or the caseous contents be too thick for aspiration, an incision may be made under strict asepsis, the abscess evacuated, the cavity curetted and treated with "Eusol" or bismuth-iodoform-paraffin paste. It is important then to close the wound tightly with mattress stitches of horsehair. In many cases it remains closed. The practice of incision and drainage with or without curetting is to be condemned, as it must be followed by infection from skin organisms.

Though Calot reports very successful results with aspiration and injection of his iodoform emulsion, in my experience it is only in the minority of cases that this procedure is efficacious in preventing the formation of a sinus. I think that radical removal before the skin is involved is much to be preferred to aspiration. Objection may be raised to the risk of bursting the abscess and spreading the caseous material all over the exposed tissues. It is almost inevitable that any large soft caseous gland will be burst in the dissection of the dense fibrous infiltration which always surrounds it; but if this be wiped away carefully at once and the cavity washed out at the completion of the operation with saline or perchloride of mercury solution, no ill effects are to be expected. It is advisable to drain for twenty-four hours through a stab puncture in such a case.

If the skin be infected and cannot be saved from breaking down, there are two lines of treatment. One is to carry out an excision, planning the skin incision so as to surround and remove the infected area of skin. The other is to treat the abscess cavity by curettage, wait till the acute inflammation has subsided and then excise the glands and communicating sinus together.

There is another type of case in which the patient presents himself with several sinuses, some healed and some discharging. Here it is better to wait if possible till all the sinuses are healed, or at least present little acute inflammatory signs; and then to excise glands, scars and sinuses in one mass, so as to leave only a linear scar. Unless the skin inflammation has disappeared the wound is apt to break down and to leave considerable disfigurement.

X-ray treatment appears to have its greatest value in such cases and discharging sinuses sometimes heal up rapidly under small fortnightly doses. I am very loath to use X-rays, however, if there is any likelihood of operation later on; for the result of the rays is to increase greatly the fibrosis around the glands and to catch up the important neighbouring structures; so that it becomes impossible to get any lines of cleavage and the difficulty and danger of operation is greatly increased. I have had no experience of massive doses, but I note that recent writers state that small doses are more suitable.

It would appear that tuberculous cervical glands constitute a much more unfavourable disease in infants. In them the course is more acute, with caseation and sinus formation and it is fairly generally held that excision is inadvisable. Some surgeons, nevertheless, practise radical excision on them. Such cases are, however, very suitable for X-ray treatment and I think that except in very well nourished and strong infants this treatment is preferable to excision. Removal of tonsils and the administration of tuberculin should be carried out as in older children.

Summary.

In conclusion the points that I should emphasize are:

(1) In a large proportion of cases moderate enlargement of the cervical glands in children is tuberculous in nature.

(2) In a certain number of cases the first manifestation of the condition is an acute abscess.

(3) The tonsil is so frequently the unrecognizable primary focus of the disease that tonsillectomy should be a routine measure in all cases.

(4) Except in infants primary excision should be carried out, unless the patient lives under good hygienic conditions and can be kept under careful observation.

(5) Caseating glands should be aspirated or incised and closed, not drained. Excision is practicable and preferable as long as the skin has not broken down.

(6) Curettage is only justifiable when a chronic sinus has formed.

(7) When there is scarring and multiple sinuses, excision will improve the appearance. This is not advisable until all sinuses be healed.

(8) Tuberculin and helio-therapy are indicated in all cases.

(9) X-ray treatment is undesirable before radical operation. It is most valuable when sinuses are present or in infants.

Dr. W. Camac Wilkinson expressed the opinion that glandular tuberculosis was in general a secondary manifestation of the disease in the cervical area and that it was usually of bovine origin.

Dr. Guy Griffiths congratulated Colonel Downes not only on his admirable presentation of his subject, but also on his degree of conservatism. It was rarely that a physician who abstained from the knife, heard a surgeon advocate other than cutting methods.

He believed that almost all cases of pulmonary tuberculosis were of human origin and that the same applied to the great majority of cases of so-called surgical tuberculosis. The usual course was that the disease was contracted by inhalation and that the bronchial lymph glands became a reservoir from which the germs were distributed to other parts of the body. The cervical glands offered the one striking exception. Here infection was often bovine through the tonsils and other upper parts of the alimentary system. He was sorry that they could not hear the subject elaborated by Dr. Camac Wilkinson. Dr. Downes suggested that tuberculosis was sometimes secondary in the glands. He, Dr. Griffiths, thought that it was usually primary and, as in the lung, the pyogenic infection followed.

In diagnosis, the von Pirquet test was of value as the patients were young and the infection recent, so that

when a reaction showed the presence of the disease, they could reasonably assume its activity and perhaps too that its site was in the glands under observation. He preferred the subcutaneous injection because it was more certain and because it was a step towards treatment.

In treatment he believed that any incision was always wrong. Even if the superficial glands were fully excised, deeper ones usually enlarged and came later towards the surface. He believed with Dr. Downes that patients sometimes recovered spontaneously; then incision was, of course, unnecessary. The treatment he adopted was general hygiene, so much easier in Australia with ample sunlight than in gloomy Europe and tuberculin. This was given not by inunction in which it was impossible accurately to regulate the dose, but by injection which was so little painful that a sleeping baby could often be injected without awakening and that children of three or four years of age soon stepped forward to receive the treatment without fear or hesitation. Aspiration was rarely required; if it were, he thought Calot's plan should be followed, though his liquefying solutions were rarely required.

Radiation too was of value, not so much radiation from the induction coil, but natural sunlight. While this might be applied in small doses directly to the lesions, it was best given to the body generally in the early morning or late evening, not in the warm climate of Australia between say 10 a.m. and 6 p.m., first to the feet and ankles, hands and wrists, later to the legs and arms and finally to the body generally, the little patient wearing only a linen hat and a loin cloth.

DYSENTERIC CONDITIONS IN CHILDREN.

By E. Marjory Little, B.Sc., M.B. (Sydney),
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and
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Between 1905 and 1914 Forsyth, (1) of Melbourne, and Litchfield Hipsley (2) and Bradley (3), of Sydney, reported the recovery of *Bacillus dysenteriae* from the stools of children suffering with so-called "summer diarrhœa." During the last three years, however, experience gained during the war in diagnosis and treatment of bacillary dysentery in adults has been applied to the civilian population in this country and the definite statement may now be made that a large proportion of children suffering from diarrhœa with blood and mucus are affected with bacillary dysentery. Comparative observations upon this disease as it occurs amongst children in three of the large cities of Australia, are now available. Since 1920 Dr. Patterson and Miss Williams (4) of the Walter and Eliza Hall Institute, Melbourne, have been investigating the problem of dysentery and the types of *Bacillus dysenteriae* isolated in Australia; Dr. Beare (5) has been inquiring into diarrhœal diseases among children in Adelaide; in Sydney during the summers of 1921-1922 and 1922-1923 the material on which the following observations have been based was collected and examined.

Although a presumptive diagnosis of bacillary dysentery may be made in adults and children suffering with acute diarrhœa associated with blood and mucus in the stools, this disease is only diagnosed with certainty by the recovery of the bacillus from the stools of such patients.

This depends primarily upon three factors.

(i.) The period of the disease at which the examination of the stool is made.

(ii.) The amount of time allowed to elapse between the collection of the material and its examination.

(iii.) The selection of material for examination.

Over the first factor the medical attendant as a rule has little control as frequently the patient is first seen late in its course of the disease. All workers emphasize the importance of examination of the stools in the early stages of the disease.

The second factor can usually be satisfactorily controlled in hospitals and institutions. In less favourable circumstances one of the two methods recommended by the Medical Research Council (6) for preserving fæces for delayed examination may be employed.

In the first the fæces are emulsified as soon as possible after evacuation with double the volume of 30% glycerine in 0.6% sodium chloride solution. In the second the fresh specimen is thoroughly mixed with an equal volume of one thirty-third normal solution of sodium hydrate.

No specimen should be left unexamined for longer than six hours.

The third factor will be dealt with later.

It has been possible to divide the cases into three main groups in each of which the clinical and bacteriological findings were similar.

Group I.

The patients were usually under twelve months, often marasmic from birth; there was a history of chronic intestinal disturbance with attacks of diarrhœa alternating with constipation, passing on to chronic diarrhœa with occasional acute periods; the stools were usually green and contained much mucus and abundant fæcal material with streaks of blood was occasionally present. These infants as a rule made little progress and the mortality rate in this series was high. From the stools of this group no pathogenic organisms were recovered.

Group II.

The patients were usually over six months old; they were acutely ill when seen with a history of sudden onset, apparently suffering from little colicky pain or tenesmus; the stools were large, liquid, offensive and seldom contained blood. These cases usually made good progress. Pathogenic organisms other than *Bacillus dysenteriae* were recovered from sixty-four cases in this group, Morgan's bacillus No. 1 and members of the Salmonella group being the most common.

Group III.

The patients' ages ranged from three months to seven years. They were previously healthy, well-nourished children. There was a history of sudden onset with diarrhœa, severe abdominal pain, tenesmus and sometimes vomiting. The children were often drowsy and sometimes showed collapse within twenty-four hours. The number of stools was always increased and almost invariably they contained

blood and mucus with little fæcal material. *Bacillus dysenteriae* was recovered from one hundred and seventeen cases in this group.

Collection of Material.

A consistent method was adopted for the collection of material. As soon as each patient was admitted to the special section the bowel was washed out with sterile normal sodium chloride solution (0.9%). The first ten cubic centimetres returned were collected in a sterile test-tube; the bulk of the fluid from the bowel was then allowed to escape and the material which was judged to be the last portion to be returned from the bowel, was also collected separately for examination. The two test tubes were dispatched to the laboratory and examined within thirty minutes. Usually the first sample was formed of fæcal material in saline solution and was often unsuitable for bacteriological examination. The second specimen as a rule consisted of flakes of mucus floating in saline solution and provided material favourable for bacteriological investigation. A higher percentage of positive findings could be expected when washed mucus was examined than resulted from inoculation with fæcal material.

Such a method is easily applied in a well-equipped hospital, but under less favourable conditions the napkin soiled by the patient should be forwarded to the bacteriologist so that he may select his own material.

Laboratory Examination of Material.

A fragment of washed mucus was rubbed over the surface of a plate of MacConkey's bile-salt medium and incubated for twenty-four hours. Colonies which failed to ferment lactose, were picked off and inoculated into tubes of glucose, mannite, dulcite, lactose and saccharose-peptone water. All were examined in twenty-four hours for acid and gas production. The lactose tubes were kept under observation for fourteen days for detection of late lactose fermenters.

In this series three types of dysentery bacilli were isolated: (i.) Members of the Flexner-Y group were recovered from eighty-six cases. These organisms were non-motile and produced acid without gas in glucose and mannite only. Agglutination tests were carried out with polyvalent therapeutic anti-dysenteric serum obtained from the Commonwealth Serum Laboratories. The eighty-six strains isolated were agglutinated by this serum in dilutions ranging from 1 in 200 to 1 in 3,200, the majority in a dilution of 1 in 800.

(ii.) Members of the Shiga group were recovered from six cases. These organisms were non-motile and produced acid without gas in glucose only. Agglutination tests were carried out with the diagnostic Shiga serum prepared by the Commonwealth Serum Laboratories with titre quoted as 1 in 1,600. All six strains isolated were agglutinated by this serum in a dilution of 1 in 800 or more.

(iii.) Late-lactose fermenters were non-motile and produced acid without gas in glucose and mannite in twenty-four hours, but left lactose unchanged for from four to fourteen days when acid was produced. These organisms were sent to Dr. Patterson and Miss Williams who were making a study of late-lactose fermenters with a view to the recognition of members of the Sonne group. Of twenty-five strains isolated in the present series twenty were agglutinated in a dilution of 1 in 1,600 or over by serum prepared at the Walter and Eliza Hall Institute with a strain of Sonne bacillus isolated in Australia.

Clinical Diagnosis of Bacillary Dysentery.

The significance of blood and mucus in the stools of patients with diarrhœal affections has been emphasized by Beare (5), Shearman (7), Wenyon and O'Connor (8), Dew and Fairley (9), who urge the importance of a provisional diagnosis of bacillary dysentery being made in such cases. The presence of mucus without blood may be regarded with less concern since practically every case of intestinal disturbance in children is liable to pass mucus in larger or smaller amounts. The presence of blood with mucus should be regarded as a sign of infection with *Bacillus dysenteriae* until proved otherwise.

In this series blood and mucus were detected by naked-eye examination in stools from one hundred and fifty-three patients. If the examination for the presence of blood had been conducted microscopically the number would certainly have been higher. *Bacillus dysenteriae* was isolated from the stools of one hundred and seventeen of these patients and 75% of these stools contained blood. Of the stools from which no pathogenic organisms were recovered, only 12% contained blood and of those from which pathogenic organisms other than *Bacillus dysenteriae* were recovered, 11% contained blood. The fact that 75% of stools from which *Bacillus dysenteriae* were isolated, contained blood in amounts visible to the naked-eye is of importance to the general practitioner and particularly to those who are not within easy access of a laboratory.

Clinically the patients with bacillary dysentery presented a striking picture and those in charge could often anticipate the bacteriological diagnosis. The patients were usually very ill, sometimes collapsed, often dehydrated and passing numerous stools containing blood and mucus. Severe abdominal pain was often present and tenesmus was a prominent feature. Intussusception is the condition most likely to be mistaken for bacillary dysentery in children and several patients admitted to the Children's Hospital were sent in with the provisional diagnosis of this condition. Careful palpation under anæsthesia, if necessary, will usually make differential diagnosis possible.

Varieties of Bacillus Dysenteriae Isolated.

Of five hundred and twenty-four cases examined in this series pathogenic organisms were recovered from one hundred and eighty-one (34%).

Of the pathogenic organisms one hundred and seventeen (64%) were *Bacillus dysenteriae.*

Bacillus dysenteriae Flexner was isolated from eighty-six cases, that is 73.5% of all cases which yielded *Bacillus dysenteriae.*

Bacillus dysenteriae Shiga were isolated from six cases, that is 5.1% of all cases which yielded *Bacillus dysenteriae.*

Late lactose fermenters were isolated from twenty-five cases, that is 21.3% of all cases which yielded *Bacillus dysenteriae.*

Serum Treatment.

It is advisable to administer anti-dysenteric serum as early as possible in the course of the disease and a polyvalent serum should be used while awaiting the bacterio-

logical report. The Commonwealth Serum Laboratories have made available a polyvalent anti-serum prepared with several strains of *Bacillus dysenteriae* Flexner, a Shiga bacillus and a Sonne bacillus all isolated in Australia. They also prepare a monovalent Shiga anti-serum for the treatment of patients from whom a Shiga bacillus is isolated.

Thirty cubic centimetres were found to be the most satisfactory initial dose of polyvalent serum. Better results were obtained with this than with smaller doses and the dose was repeated daily for five days even when rapid improvement followed the first administration. The illness was of shorter duration in cases in which this procedure was adopted. In the first cases the serum was administered intramuscularly, but this method was sometimes followed by distress and prostration on the part of the patient and in dehydrated infants it was at times found difficult to give the full amount of serum on several successive days. Later Dr. Margaret Harper initiated the practice of administering the serum intra-peritoneally and this method has been followed by gratifying results. The thirty cubic centimetres of serum are added to one hundred and fifty cubic centimetres of a solution of 5% glucose in normal saline solution, so that serum, food and fluid are administered together. The only apparatus required is a funnel, length of rubber-tubing and paracentesis needle. The fluid is warmed to body temperature and allowed to gravitate through the needle into the peritoneal cavity of the child. Very little discomfort attends this method of administration, the patient usually lying quite still.

Of eighty patients in this series who received serum, eighteen died, that is 22%. Of thirty-one patients who did not receive serum, thirteen died, that is 41.9%. Of the patients who died after receiving serum, three had Shiga infections, one was four months old and weighed two and a quarter kilograms (five pounds), one received serum on the fifty-sixth day of illness, two on the twenty-first, one on the sixteenth, one on the fourteenth and two on the twelfth. Only four received serum within four days of the onset of illness. Of the patients who were treated with serum and recovered, 85% received serum within a week of the onset of illness.

Summary.

(1) In a series of five hundred and twenty-four cases of diarrhœal affections in children pathogenic organisms were recovered from 34%.

(2) Of the pathogenic organisms isolated 64% were *Bacillus dysenteriae*.

(3) The Flexner type of *Bacillus dysenteriae* was recovered in 73.5%, the Shiga type in 5.1% and the Sonne type in 21.3%.

(4) Bacillary dysentery in children is readily recognized clinically. The chief features are the acute onset with diarrhœa characterized by blood and mucus, severe abdominal pain and tenesmus.

(5) Early administration of anti-dysenteric serum should be adopted and the intra-peritoneal route is recommended.

Acknowledgements.

Our thanks are due to the members of the honorary medical staff of the Royal Alexandra Hospital for Children for permission to investigate the cases under their care; to Dr. Mona Ross, Dr. Williams and Dr. Durie for valuable assistance in the work of agglutinating strains and to Sister Tanner and Sister Coggins for their willing cooperation.

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DYSENTERIC CONDITIONS IN CHILDREN.

By F. H. BEARE, M.D., B.S. (Adelaide),
Mile End, South Australia.

IN view of the fact that unfortunately I am unable to be present in person to join in this discussion, I have thought it best merely to state facts observed by myself and leave the meeting to draw whatever conclusions they may think fit from them.

During the years 1920 to 1922 I was engaged on work for the Gastro-enteritis Research Committee of Adelaide and it is with the permission of this Committee that I contribute the following data to the discussion.

As there is a limit to the time allowed to each speaker in the discussion I have confined my remarks to those aspects of the subject that to me have seemed the most important.

First, it would seem, if one can take the official statistics in the Commonwealth Year Book as a guide, that the true importance of dysentery as a cause of death has not been fully recognized: and this applies to adults as well as children. It is my opinion that in the course of time when the wide-spread incidence of bacillary dysentery becomes more recognized, this infection will rank as one of the first three most important causes of death in children under the age of two years.

Type of Infection.

In the Adelaide series there was no instance of amœbic infection found. Of the bacillary type, no infection with Shiga's organism was found, and in every case except one in which the infecting organism was isolated, Flexner's, organism was present. The remaining case was due to infection with Sonne's bacillus.

Various observers have shown that in any case of clinical bacillary dysentery the likelihood of isolating a dysentery organism decreases with the time after the onset the stool is examined. In the Adelaide series the percentage of positive findings at any one period after the onset of the illness was, in my opinion, quite high enough to support the thesis that "every case of diarrhœa characterized by the passage of blood and mucus in the stools should be regarded as bacillary dysentery unless proved to be some other condition." Flexner's bacillus was not found in either the stools of normal children nor in those of children suffering from diarrhœa not containing blood mucus. Of the former, seventy-five specimens from fifty-three children did not contain dysentery bacilli; of the latter, in over three hundred specimens from one hundred and thirty patients no dysentery bacilli were found, except one specimen (the case from which this specimen was derived was doubtful as regards history previous to admission to hospital).

Age is a definite factor in the ætiology. Thus of all children under three months of age with diarrhœa examined 10% contained dysentery bacilli, 37%, 48% and 48% respectively in each successive trimester of the first year. In the case of children over one year of age 64% were suffering from dysentery.

Diet plays an important part. A child on the breast is much less likely to be attacked by dysentery than one artificially fed. As regards the variety of artificial feeding, it would seem that it mattered little with what particular food the child was fed as far as the risk of a dysentery infection was concerned. As cow's milk is the commonest form of artificial food in general use at all ages, more children develope dysentery on this food than on any other.

Infectiousness.

Dysentery is infectious and communicable, for of fifty-three patients from whom records could be obtained, thirty-one or 61% had been in contact with another patient either in the house or next door. Surely there are none of the notifiable diseases that can show such a high percentage of associated cases.

Horses and Flies.

There is a close association between flies and horses and the incidence of dysentery; probably the association with the horse is explained by the fact that flies breed freely in horse manure. Thus in fifty-one homes from which dysentery patients came, a horse or horses were in association in forty-one *id est* 80%. In three hundred and seventy-six homes from which non-dysenteric patients came, the association was only 39%.

Of fifty-two homes from which dysentery patients came, flies were very prevalent in forty-two.

Carriers.

Several instances in which the Flexner organism was isolated from a healthy adult who apparently infected a child or a group of children, were observed.

THE RESULTS OF THE SERUM TREATMENT OF BACILLARY DYSENTERY IN CHILDREN.

By Reginald Webster, M.D. (Melbourne),
Pathologist to the Children's Hospital, Melbourne.

The children included in this series (66) were subjected to a rigid bacteriological control. Fifty-seven (57) were infected with *Bacillus dysenteriae* (Flexner) and nine (9) with the Sonne bacillus, which is a recognized cause of dysentery and is closely allied to "Flexner" group.

No child is included in whom the organism isolated failed to exhibit and maintain for three weeks in sugar-media the characteristic reactions of the Flexner or Sonne dysentery bacilli or failed to agglutinate in good titre with the appropriate serum.

Table I.—Result of Treatment of Bacillary Dysentery in Children by the Administration of anti-dysenteric serum; Children's Hospital, Melbourne, 1922-1923.

Age Group	Serum	Number of Patients Treated	Recovered	Died	Mortality
					%
I. Under twelve months	None	17	8	9	52.9%
	Given	16	5	11	68.7%
II. Twelve months to two years	None	10	4	6	61%
	Given	12	1	11	91.6%
III. Over two years	None	10	10	0	0
	Given	1	1	0	0

Total number of children considered 66
Gross mortality .. .. 56. %
Total mortality in the non-serum treated (15 of 36) 40.5%
Total mortality in the serum treated (22 of 29) 75.8%
Twenty nine children received serum. ..

Table II.—Results of Serum Treatment according to the day of illness at the time of the first injection.

Day when First Injection was Given	Number Treated	Recovered	Died
Second	1	1	0
Fourth	5	1	4
Fifth	3	2	1
Sixth	3	0	3
Seventh	4	1	3
Eighth	3	1	2
Ninth	3	0	3
Eleventh	5	1	4
Seventeenth	1	0	1
Unknown	1	0	1

It is important, however, to take into consideration the stage of the disease at which serum was first given and it is fair to consider all treated with serum later than the fifth day as non-serum treated.

Table III.—Results of serum injected on or before the fifth day.

First Injection of Serum	Number Treated	Recovered	Died	Mortality
On or before fifth day	9	4	5	55.5%
After fifth day or none	57	25	32	56.1%

It has been the experience at the Children's Hospital that children who contract dysentery due to *Bacillus dysenteriae* (Flexner) when they have passed the age of infancy almost invariably do well irrespective of serum treatment. This is illustrated by Group III. In an attempt to assess the value of anti-dysenteric serum, it gives fairer consideration to the serum if Group III. be omitted, for of the eleven in this group ten received no serum. These children were bound to do well and it is apparent that the inclusion of the group works to the disadvantage of the serum by lowering the mortality rate of the non-serum-treated series.

It is to be noted, however, that in the figures published by Dr. Beare, of Adelaide, children of four and five years of age are included.

In other words if one wanted to make out a case against the use of anti-dysenteric serum the inclusion of Group III. (children over two years) would be of great assistance. If Group III. be omitted, the mortality in the non-serum-treated series rises from 40.5% to 55.5%.

Perhaps the fairest presentation of the results is embodied in Table IV. in which all who received the first injection later than the fifth day are regarded as non-serum-treated and Group III. are omitted.

Table IV.—Results of Serum Treatment in Children not over two Years.

First Injection of Serum	Number Treated	Recovered	Died	Mortliaty
On or before fifth day	9	4	5	55.5%
Later than fifth day	47	15	32	68%

Average quantity of serum given .. .. 115 c.c.m.
Unusual injection .. .. 30 c.c.m. to 60 c.c.m.
Smallest single dose employed .. .. 20 c.c.m.
Largest single dose employed .. .. .. 250 c.c.m.

The serum was administered by the subcutaneous and intra-peritoneal routes.

Dr. S. W. Ferguson said that every form of diarrhœa in infancy had its own definite characteristics. In diarrhœa depending on dietetic causes, such as excess of fat, sugar or protein, the motions exhibited distinctive features which were indicative of the underlying dietetic error. In each there was a change in the intestinal flora. In the essentially infective diarrhœa there was invasion by a definite and usually specific organism. In both the dietetic and infective types treatment must be conducted with regard to the nature of the intestinal bacterial flora. In bacillary dysentery of infancy and childhood a diet rich in carbo-hydrate had been shown to be inimical to the activities of the infecting organisms. As much fluid as possible should be given in the early stages, carbo-hydrates should be gradually replaced by protein and fats should be withheld until last. In general Carnrick's soluble food gave excellent results. He frequently ordered small doses of castor oil to which were added one or two drops of some opiate for the purpose of allaying the irritation. He expressed great disappointment in serum therapy.

Dr. T. E. Green advocated lavage of the colon as an additional therapeutic measure.

Dr. H. Douglas Stephens asked if any of the members of the Section had had experience of colitis caused by the "gas bacillus." He questioned whether the intestinal flora were ever altered by dietetic measures; whatever was done in this direction the organisms would still be living in a protein medium. He was inclined to the use of diluted sterile milk ("Bengerized") in small quantities early in the course of the disease and as soon as the high fever subsided.

Dr. Margaret Harper endorsed the remarks of Dr. Stephens with regard to dieteties.

Dr. W. H. Fitchett drew attention to the necessity for maintaining a sufficient caloric value in the diet.

Dr. Vera Scantlebury advocated the use of sodium sulphate in large doses during the first two days of the disease provided there was no great degree of dehydration.

Dr. Marjory Little urged that further work should be done in the direction of collecting data dealing with the results of serum therapy in dysenteric infections of infancy and childhood.

THE PROBLEM OF RICKETS IN AUSTRALIA.

By E. J. Dalyell, O.B.E., M.B. (Sydney),
Sydney.

The question of rickets has not attracted much attention in this country where severe grades of the disease are rarely seen and the occurrence of the milder form has been little considered. In the light of the results of investigation elsewhere a review of the subject at this time is opportune, in order to determine the application of our present knowledge to Australian conditions.

Recent researches have led not only to exact information regarding the cause and prevention of this disease, but have established also new principles relative to the metabolism of inorganic constituents within the body which are likely to have a profound effect on our ideas of cell nutrition in general. These new principles have reference chiefly to the specific effect of light rays of certain wave-length on the metabolism of calcium and phosphorus and to the interaction of radiant energy with particular dietetic elements in regulating the metabolism of these inorganic substances. A new field of inquiry has been opened up by recognition of the specificity of light stimuli in metabolic processes, a conception which may be applicable to organic as well as to inorganic constituents of the body.

At the outset it is desirable to define what is meant by rickets, but a precise definition has become more difficult as our knowledge of the subject has increased. The accepted clinical description of bone deformities and other characteristic stigmata refer to the late manifestations of the disease and simply indicate the results of long-standing disorder of the metabolism of calcium. In earlier

stages slight abnormalities of the bones of head and thorax may be detected clinically and defective formation of long bones shown by careful radiographic examination, but the initial changes are indefinite and difficult to recognize with certainty. Although the fundamental metabolic disturbance in rickets is not yet fully understood, much light has been thrown on it by bio-chemical investigation. This shows that in rickets the normal concentration of calcium and of inorganic phosphorus in the blood is altered and that the characteristic change that occurs is a reduction in the amount of inorganic phosphorus, the amount of calcium being within normal limits. When reduction in the amount of calcium occurs, it is associated with symptoms of tetany in rhachitic children and the amount of phosphorus may not be greatly reduced. The effect of specific anti-rhachitic therapy is to restore phosphorus to a normal level in the blood and this adjustment is accompanied by the deposition of calcium in bony tissues. McCollum and his co-workers in Baltimore who have studied the question exhaustively, describe two forms of rickets, calcium-low and phosphorus-low rickets, and they emphasize the fact that variation from the normal ratio of calcium to inorganic phosphorus in the blood is an integral part of the rhachitic condition. This altered concentration in the blood is convincing evidence of the generalized disturbance that occurs in rickets and the condition is to be considered as one of incomplete calcium and phosphorus metabolism leading to defective bone formation and probably affecting all the body tissues in some degree.

The agents concerned in normal growth and development of bone are (i.) radiant energy, (ii.) an organic dietetic factor, (iii.) the supply of calcium and phosphorus in the diet. All are closely interdependent and the reciprocal action of the first two is so intimate that neither of them can be considered apart from the other. In the human organism radiant energy appears to be of fundamental importance and the biological power possessed by light waves is the most suggestive fact that has emerged from the study of rickets.

The light rays with specific effect on calcium and phosphorus metabolism are ultra-violet radiations with a wavelength of about 300 micro-millimetres. Their distribution in the spectrum of sunlight is limited to a small region at the end of the visible solar spectrum and their intensity in sunlight varies with season, with time of day and with the density of the atmosphere. The most important practical point regarding the rays is that they do not penetrate ordinary window glass and therefore brilliant sunlight coming through glass windows fails to provide the rays essential for promoting calcium metabolism and preventing rickets. This explains the high incidence of rickets in northern climates during the winter when exposure to direct sunlight is at a minimum. In the clinical investigations carried out in Vienna in the last three years it was shown conclusively that exposure to sunlight without other dietetic or environmental change initiated the process of calcification in bones of rhachitic infants who had developed the condition during the winter while living in hospital under excellent hygienic conditions. Other sources of light than sunlight have been shown to produce ultra-violet radiations in much greater concentration than occurs in sunlight. The mercury vapour quartz lamp, the cadmium spark lamp and the carbon arc lamp have all been successfully used for the prevention and cure of rickets by light therapy.

As already stated an organic dietetic factor has a close relation to radiant energy in stimulating normal calcium metabolism. The factor has not been isolated, but its distribution in various food stuffs has been studied by animal experiments. It occurs in close association with the fat-soluble A vitamin, the accessory factor essential for promoting growth and preventing xerophthalmia; the two, however, are not identical, as the action of the calcium-depositing factor can be demonstrated apart from that of the fat-soluble growth factor in the same food-stuffs. Both vitamin A and the calcium-depositing factor occur most abundantly in cod liver oil and in the liver oil of other deep sea fish; egg-yolk is also a rich source of both factors. Human milk and cow's milk contain the growth factor in lower concentration and so far as is known at present, these important food-stuffs supply relatively small amounts of the calcium-depositing factor. The interaction of this factor with the specific ultra-violet rays, however, permits one to replace the other within wide limits. When the intensity of ultra-violet radiation is great as in summer, a diet in which milk is the only source of the calcium-depositing factor is adequate for prevention of rickets. In winter rickets may develope on a similar diet, if direct exposure to sunlight or other source of ultra-violet rays is not possible. Artificial radiation by mercury vapour or other lamps can prevent developement of rickets by increasing the intensity of the light agent; or further concentration of the calcium-depositing factor can protect from rickets without resorting to additional ultra-violet radiation. In preventing or treating rickets in infants the only means of increasing the calcium-depositing factor adequately has been by the addition of cod-liver oil. Egg yolk has been used prophylactically, but not therapeutically. In Vienna and in American cities where careful observations have been made administration of large amounts of human milk or cow's milk alone has not been effective in preventing rickets under winter conditions.

Both ultra-violet light and the concentrated calcium depositing factor act similarly. The effect of these dissimilar agents is to adjust the concentration of calcium and phosphorus in the blood and as the total amount and relative proportion of these substances are maintained at the normal level, bone formation proceeds normally and rhachitic areas of defective bone are replaced by healthy osseous tissues. This adjustment can take place without any increase in the amount of calcium or phosphorus ingested and it has been shown repeatedly that the supply of these salts ingested during a rhachitic period is adequate for the needs of the organism if the stimulus of the light and dietetic factors is supplied. It would seem that these agents in some way activate the processes of absorption, assimilation and utilization of calcium and phosphorus and the metabolism of the elements cannot continue when the combined action of light and diet falls below a certain degree of potency. The extraordinary fact that the same effect is produced on the metabolism of calcium and phosphorus by adding cod-liver oil to the dietary as is obtained by exposing the surface of the

body to light rays of a special wave-length is at present unexplained. The fact that the cod is a deep water fish and must have a metabolism adjusted for absence of sunlight is a possible clue to this strange interaction, but such is merely hypothesis and much investigation is needed before the obscure fact is understood.

One other important point regarding light must be outlined. It has been shown by animal experiment that the solar spectrum contains rays which appear to counterbalance the action of the ultra-violet wave lengths. It is possible to inhibit the beneficial effects of ultra-violet radiation on animals receiving a rickets-producing diet by exposing them at the same time to infra-red rays of long wave-length, a fact for which we have at present no explanation, but which emphasizes the profound influence to be attributed to the light factor.

The application of these principles in Australia calls for much investigation which should prove interesting and profitable. The first question that arises, is the degree of rickets that occurs in Australia. Gross stigmata are admittedly rare, but minor grades of rickets are not infrequent and the degree to which they occur, should be determined accurately. The most suggestive evidence of disordered calcium metabolism is provided by the prevalent defects in teeth in this population. Bearing in mind the chemical evidence of disturbed calcium metabolism, data should be sought regarding the normal level of calcium and phosphorus in the blood in infancy. When abnormality occurs, its cause may be pre-natal or due to insufficient exposure to light or to inadequate supply of necessary elements in food.

Analysis of sunlight to determine the intensity and penetration of the ultra-violet rays is essential and study of light effects in this country should be coordinated with the intensive work on the subject now proceeding in England.

The food factors demand close attention in this country. Light conditions are so good that it is natural to suspect dietetic defects as chief contributors to such stigmata as occur, more particularly to dental defects. The organic factors in ordinary diet of infancy is probably adequate, but accurate investigation of this vitamin in relation to calcium and phosphorus supply is essential. This is particularly indicated in areas in which cattle are known to suffer from bone defects as is recognized in some districts.

In conclusion the object of this brief outline is to present for your consideration the new principle of light effects on the body metabolism and to emphasize our responsibility for understanding the necessary conditions for proper growth and developement of bone, so that errors which are preventible, may be eliminated.

RICKETS.

By H. M. Mayo, M.B., B.S. (Adelaide),
Honorary Physician to Out-Patients, The Adelaide Children's Hospital.

For a clinician like myself, my duty in such a discussion is to bring forward data concerning cases, so as to serve as a basis for conclusion.

The first questions to ask are:

(1) Does rickets occur in Australia?
(2) If it does, under what conditions does it develope?
(3) What is its extent?

Does Rickets Occur?

I have looked up the records of the Adelaide Childrens' Hospital since 1895 and have found some forty children classified as suffering from rickets.

On going through these case sheets I have selected twenty-three as possessing evidence from the records of a disease which might be rickets.

Some of the other cases were almost certainly rickets without sufficient data recorded to be conclusive, while others were definitely scurvy.

These twenty-three cases were for most part in children from eighteen months to three years with definite bowing of the legs, in some cases extreme. Eight were said to have beading ribs. Harrison's sulcus was present in one, and a violin thorax in one. An open fontanelle was mentioned in two children, both over two years, and seven were stated to have bossed heads.

Cranio-tabes was mentioned as absent in two, otherwise not recorded. Sweating of the head was recorded in six. There was a prominent abdomen in four. One child, aged two and a half years, had been breast-fed for one week and had had condensed milk and other foods after. This child born in October, showed bossed head, enlarged epiphysis of all the long bones, bowing of the femora and tibiæ and a violin thorax; when sitting she propped herself with her hands. This was one of the worst cases recorded.

The feeding in six children was not mentioned.

In eleven breast feeding was maintained for a considerable period, seven for about a year or even longer.

There were two January babies, one February baby, one March baby, three April babies, one June baby, one July baby, four August babies, three October babies, three November babies, and four December babies.

These figures seem to show that rickets do occur, though probably infrequently to such an extent as above.

These children came from country and town in varying proportion and in such small numbers that the conditions must be dependent on special circumstances.

One child I know lived in a house in the Mount Lofty Ranges in a small house right on the road without verandah. The winter season is very wet in the hills. The child may have been and probably was kept in doors for long periods.

I can quote also from my personal observation the cases of three children similar to the above though not in degree, who showed, sweating heads, beading of ribs, prominent abdomen, some bossing of head and enlarged epiphysis, whose history showed that two had been kept indoors for months, because they were good children and sat quiet and had "bronchitis."

One of these children was in a very good home and was breast fed though somewhat inadequately for six months. She gained very slowly about fifty grammes a week; at six months her diet was supplemented and some two months later she showed signs of rickets, sufficient to cause disquietude.

She has now been treated and is improving rapidly.

Of the other two one was breast fed for a time, very inadequately also and then had considerable digestive difficulties.

The other was given everything and lived in a most unhygienic home.

These two we know from the visits of the welfare nurses were kept indoors and great benefit began as soon as they were put outside.

I think I have shown that cases of rickets do occur in South Australia of sufficiently severe type to cause disabling deformity.

The next question is to what extent is rickets prevalent.

Here I can give no satisfactory answer. Dr. Gertrude Halley, our one School Medical Officer for 80,000 children, could not without great increase of staff make anything like a comprehensive survey.

She tells me that among school children she has never seen really severe rickets, though she is familiar with minor degrees. Minor degrees are found, however, both in Adelaide and in the larger mining centres in the country. In my own experience it is rare to find major rickets, perhaps partly because I am a physician and the worst deformities go to the surgeon.

I must say, however, that I see what seems to me a very large proportion of minor rickets among the many hundred children who pass through my hands in the year.

Among the records of three hundred and forty-two children under the care of the welfare centres which were classified for the breast feeding, there were two with rickets as far as I know. These showed definite signs. Two of those mentioned above were in this series. This is by no means a sure criterion of the real number of cases, as minor degrees easily pass unrecognized in the early stages. The fact that the mothers were regular in their attendance was a sign in many cases of a willingness to be guided and to obey rules of hygiene.

The Matron at the School for Mothers has told me that among the children visiting the school there are certainly more with what I call minor rickets than she remembers some eight or nine years ago. Whether this is due to a better recognition or to more frequent occurrence, she cannot say.

The nurses, however, tell me that in the last six years overcrowding has become increasingly prevalent.

It frequently happens that the owner of a house will keep the ground floor and let the upper rooms of the house. A whole family may inhabit a room and if without a balcony, it becomes impossible for the mother to have the children out of doors at all, except when she takes them out. This leads to a young baby being continuously indoors and with such overcrowding to conditions that are most undesirable.

With regard to minor rickets I find that chest deformities are common, probably owing to two factors. One is the comparatively short winter followed by the very sunny summer and the other is the great prevalence of coughs and colds during the winter and also in other seasons.

In short winter is very apt to combine with respiratory infections to keep the children indoors, but when the warm days come, if it is by any means possible, they will be out and so the harm done by the darkness is partly counteracted, the chest deformity often being the only noticeable residual sign of trouble. As soon as children can walk, they will be outside, so that if locomotion is not interfered with, improvement is probably rapid.

In my experience besides chest deformities, head sweating is a common symptom. Irregular dentition and protuberant-abdomen, though not quite certain signs, are suggestive.

Bossy head is often seen, but cranio-tabes is conspicuous by its absence or at any rate infrequent occurrence.

As far as feeding is concerned, a number of children who developed rickets were breast-fed, sometimes apparently contented, satisfied children, sometimes not.

Quite a number have been breast-fed unduly long. This I regard as more probably an indication of poor hygiene and poor intelligence than necessarily a food deficiency problem, as the breast milk is often supplemented by a varied diet.

In one infant which was under my care at the Babies' Hospital the feeding was so difficult that it is quite probable that food deficiency was a factor. He was one of those miserable atrophic babies who lay with retracted head and flexed limbs. He subsequently did very well, but developed after beginning to gain in weight, sweating of the head and beaded ribs and now shows a very ugly chest deformity and some bossing of the head.

RICKETS.

By Margaret Harper, M.B., Ch.M. (Sydney),
Honorary Physician, Royal Alexandra Hospital for Children, Sydney.

During the months of July, August and September of 1923 a series of infants between the ages of one month and two years was examined for evidence of rickets.

The total number examined was one hundred and eleven and of these fourteen showed definite signs of rickets, *id est* 12.3%.

Enlarged costo-chondral junctions with failure of normal closing of the fontanelle were regarded as signs of rickets. In most cases there were other signs as well, such as some thoracic deformity, enlarged epiphyses, muscular hypotonia, sweating and delayed dentition.

The fact that beading of the ribs may be caused by scurvy, was borne in mind and care was taken to exclude this disease. None of these infants had any symptoms of s urvy, florid or latent, and in no case did an X-ray examination show any signs of scurvy. Most of the infants received orange juice. The quantities varied and in some cases it was not given regularly.

Age.

Thirty-nine infants under six months of age were examined and of these only two showed signs of rickets. Both were four months old. One of these was breast-fed for two weeks and the other artificially fed from birth.

J.M. was a premature infant weighing two and a half kilograms (five and a half pounds) at birth. His mother suffered from puerperal insanity and was in a mental hos-

pital. His head was square with frontal bosses and a widely open fontanelle. The beading of the ribs was very marked. Harrison's sulcus was present and slight muscular hypotonia. His diet when he came under observation was full-strength dried milk. He was kept in the house all day, except for short periods when he was carried out by his guardian.

W.W., the other four months' old infant, was put to the breast once every night for one week and thereafter had a variety of food, such as dried milk, condensed milk and "Benger's Food." He had a large anterior fontanelle, with some cranio-tabes along the occipito-parietal suture, enlarged costo-chondral junctions, flaring of the ribs and enlarged epiphyses. He also was in the house all day, except for the periods when he was carried out by his mother.

The ages of the other rhachitic infants were: two of six months, two of seven to seven and a half months, one of eight months, one of nine months, one of ten months and four between twelve and eighteen months.

These are the ages at which the infant came under observation, not the age at which rickets began.

Fresh Air and Sunlight.

Most of these babies were said to be out of doors nearly all day, some on a verandah or balcony, others in a yard or garden. The two already mentioned and one other were the only ones known to be kept most of the day indoors.

Diet of the Rhachitic Infants.

Dried milk was the diet of nine babies, two had sweetened condensed milk as well. Sweetened condensed milk was the diet of three, one had butter added, but the quantity is not known. Fresh cow's milk diluted with biscuit added was the diet of one. Humanized milk with "New Zealand Emulsion" was the food of one.

One of the infants fed on dried milk was reported to have had cod liver oil, but the quantity given was not known. No breast-fed baby in this series showed signs of rickets.

Intercurrent Illness.

Two of these infants had had enteritis. One had been in hospital three months. One suffered from habitual vomiting. One had had pneumonia, measles and enteritis and had been in hospital for seven months out of his first year. None of the other ten had had any illness.

The diet of the ninety-seven infants showing no signs of rickets, was entirely breast-fed in sixty-nine or 62%, dried milk in twenty-two (three with the addition of cod liver oil) or 28%, condensed milk in four or 6.4% and fresh cow's milk in two or 3.6%, one with the addition of cod liver oil.

It is impossible to draw conclusions from such a small number of cases when the hygienic conditions and details of feeding are not exactly known. One fact, however, stands out prominently, that no breast-fed infant in this series had rickets.

It was necessary to depend entirely on the statements of the mother or guardian with regard to the amount of sunlight and fresh air these infants had. But, considering our climate, except in the three cases already mentioned, it does not seem likely that this was deficient. In Sydney it is impossible in a large number of cases to use fresh cow's milk as food for infants. We have nothing to compare with the Talbot and Wilsmere milk supplies. Hence the dried milks are used to a very large extent. Many of the infants who are under the supervision at the Baby Health Centres, receive in addition to modified milk (fresh or dried) a 50% emulsion of cod liver oil.

The severest case of rickets occurred in an infant born in July, 1922, in England. This infant was under dietetic treatment in the Babies of the Empire Mothercraft Centre for four months. Its food was humanized milk with "New Zealand Emulsion." From the history given by the mother it was in a state of severe malnutrition when admitted. The infant was in the home in London during winter months and consequently cannot have had much sunlight. The "New Zealand Emulsion," is said to contain 33% of animal fat, of which only a fraction is cod-liver oil. From the researches of Dr. Dalyell and her colleagues and others it is evident that no fat except the liver fat of other deep sea fish can take the place of cod-liver oil in the prophylaxis and cure of rickets.

McCollum and his collaborators have shown that in animal experiments the "amounts of calcium and phosphorus in the diet and also the proportions between these two elements exert a profound influence in bone growth."

It has been proved that the percentage retention of calcium is much lower on a diet of cow's milk than on one of human milk. Hence with dilute milk mixtures, although the amount of calcium present may be equal to or even greater than that in human milk, the calcium retention may fall below the amount required, especially when, as in this case, neither sunlight nor cod-liver oil are obtained in sufficient quantity to enable the infant to make the most economical use of the calcium available. The changes which take place during the process of manufacture in the dried milks, are not thoroughly known. But it is possible that these may be such as to render the infants fed on them more liable to develope rickets.